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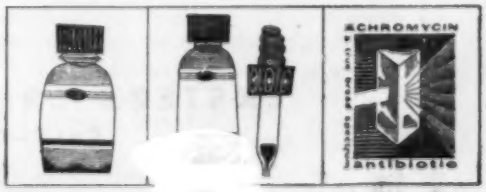

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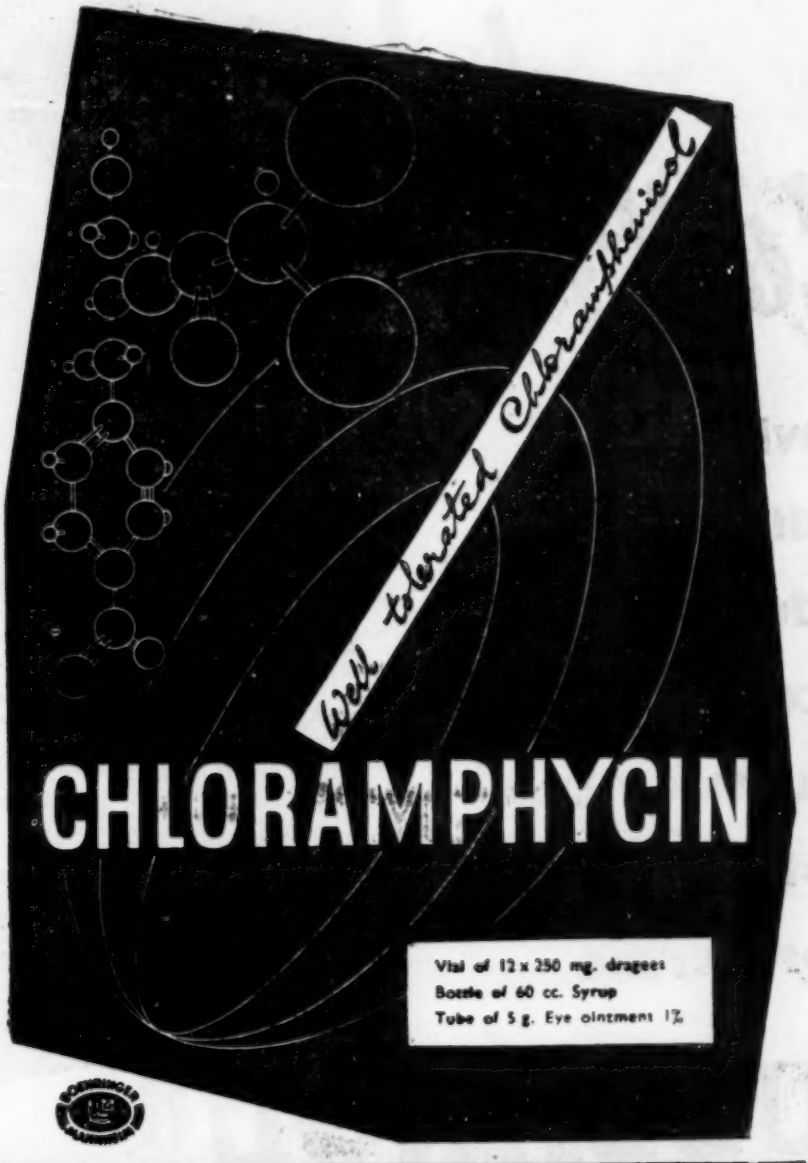

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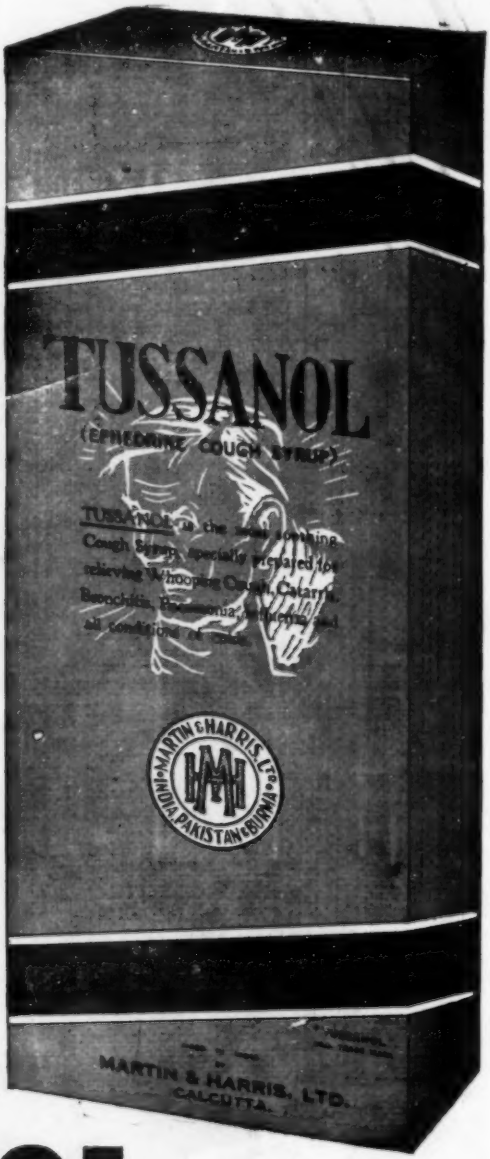

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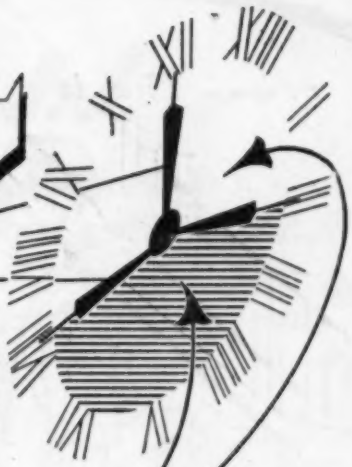

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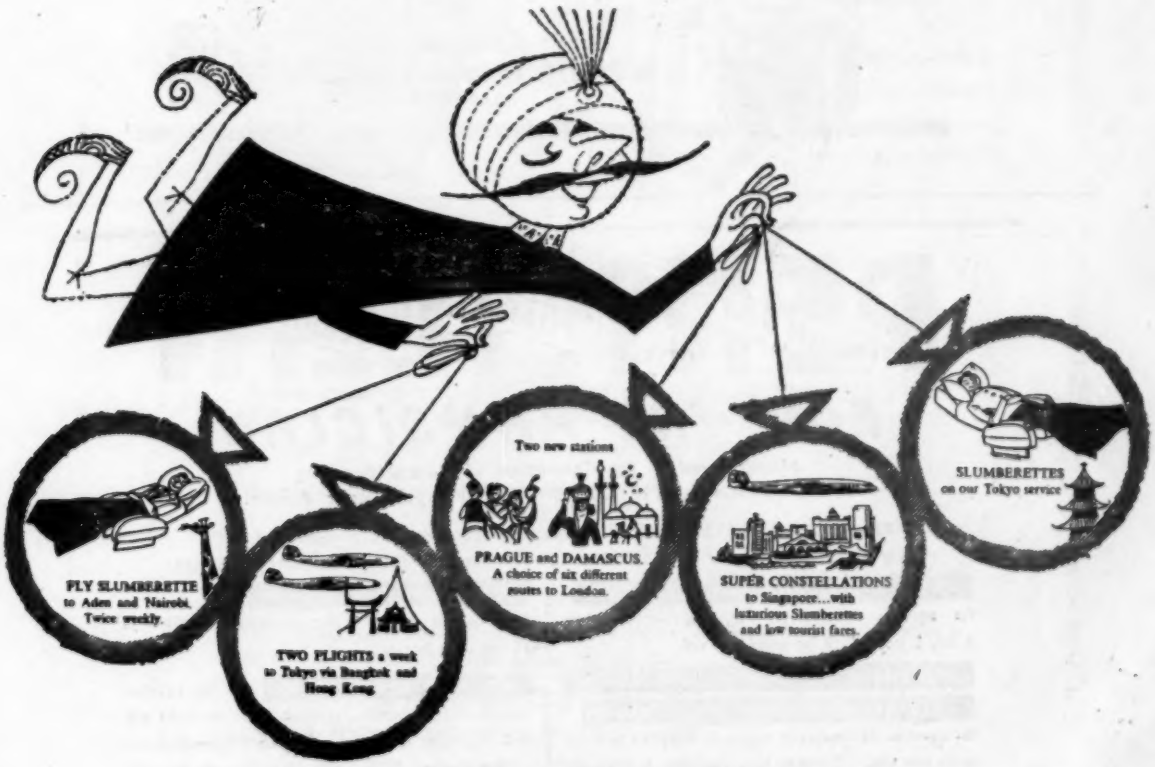
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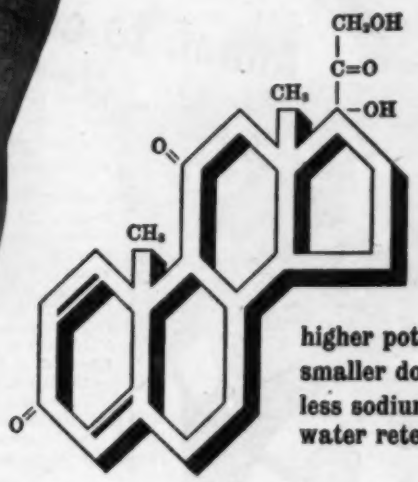

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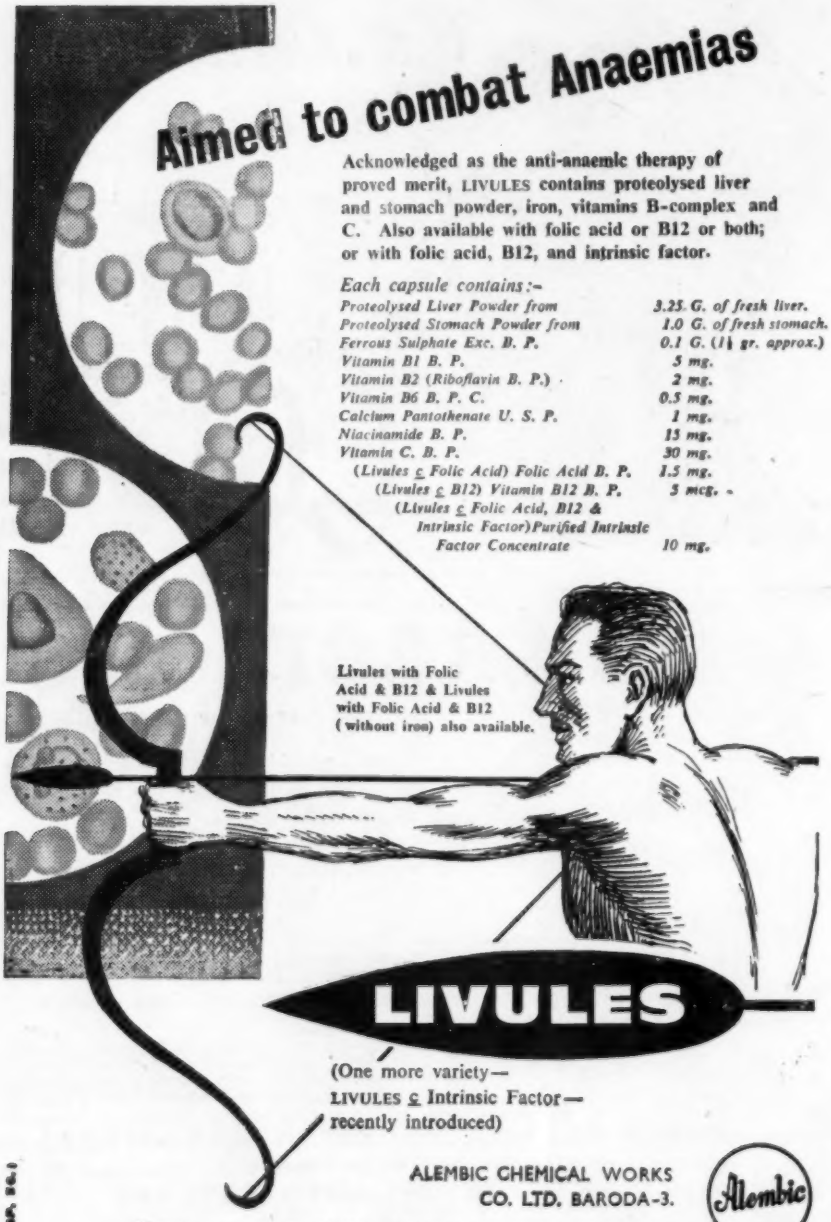

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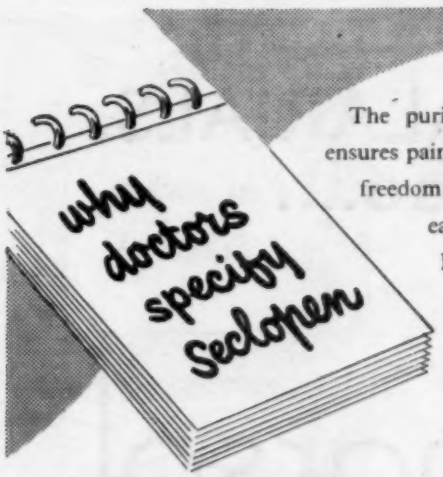

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ORIGINAL ARTICLES

PATTERN OF LIVER DISEASES IN RAJASTHAN

(An Analysis of Histopathological Examinations of 401 Livers from Biopsy and Autopsy Cases)

R. K. GOYAL, D.SC., PH.D., M.R.C.P., F.R.S.E., M.R.C.S.

AND

J. C. SARIN, M.B.B.S., D.T.M.,

Department of Pathology, S. M. S. Medical College, Jaipur.

During the last three years 1952-1955, we had an opportunity to study 331 specimens of liver biopsies and 76 livers from autopsies conducted in the pathology department of S. M. S. Medical College, Jaipur.

An analysis of the results of this examination provides interesting data pertaining to some lesions of the liver. The following few paragraphs sum up our findings in this regard.

Cirrhosis of the Liver

There are conflicting reports in the literature regarding the relative incidence of the 'postnecrotic' variety and the 'diffuse hepatic fibrosis' variety of cirrhosis. The former is the sequela of necrosis usually caused by infective or toxic agents which may or may not be associated with dietetic deficiency and the latter variety of cirrhosis is considered to be due to deficiency in certain dietary factors. The histological features of the advanced type of cirrhosis offer no differentiating factors between the two types of cirrhosis. However, large bands of fibrous tissue, mononuclear infiltration and absence of fatty infiltration are said to characterise postnecrotic cirrhosis while in contrast 'diffuse hepatic fibrosis' is likely to show fatty infiltration, fine strands of fibrous tissue and polymorphonuclear infiltration (Litchman, 1949; Spellberg, 1954). Judged by these criteria various authors, both abroad and in this country, have published papers showing the relative frequency of these two types of cirrhosis.

From the material available, there were 40 cases of cirrhosis of the liver. Of these 40 cases, 21 cases when judged by the criteria stated above would be classified as 'postnecrotic cirrhosis'—these showed areas of focal necrosis, relatively thick bands of fibrous tissue, mononuclear infiltration and no fatty infiltration. Out of the remaining 19 livers, 11 showed fatty infiltration, along with irregularly disposed fine strands of fibrous tissue, and two livers specimen showed in addition to these features—scanty areas of focal necrosis. These 13 cases could, therefore, be classified as 'diffuse hepatic fibrosis'. Three of the remaining livers showed extensive irregularly disposed fibrous tissue hyperplasia and marked reticulo-endothelial hyperplasia. The reticuloendothelial cells in these cases were full of malarial pigment. These were examples of 'malarial cirrhosis'

(Spellberg, 1954). Another case showed a 'diffuse-hepatic fibrosis' variety of cirrhosis along with typical tuberculous lesions in the liver.

In addition to the above mentioned 38 cases of cirrhosis, two cases dying of cardiac failure showed an extensive fibrous tissue hyperplasia in the liver which could be considered consistent with a diagnosis of cardiac fibrosis.

TABLE 1—SHOWING RELATIVE INCIDENCE OF VARIOUS TYPES OF CIRRHOSIS IN 331 BIOPSIES AND 76 AUTOPSIES

Type of cirrhosis	No. of cases		Percentage of total
Postnecrotic	21		52·5
Showing both fatty degeneration and focal necrosis ...	2		5
Diffuse hepatic fibrosis ...	11	12	30
do. with coexistent tuberculosis liver	1		
Malarial cirrhosis	3		7·5
Cardiac cirrhosis	2		5

INFANTILE CIRRHOSIS

Another problem in diseases of the liver is the syndrome termed infantile cirrhosis. Of ill-understood aetiology, the disease is characterised by a definite clinical picture. A recent review on the subject, published by the liver sub-committee under the auspices of Indian Council of Medical Research (1955) lists the following as prominent features of this entity—areas of necrosis, irregular fibrous tissue hyperplasia, bile duct proliferation and cellular infiltration. Fatty infiltration is reported to be infrequent.

We examined livers from 4 cases which were diagnosed as infantile cirrhosis. The clinical symptoms in some cases were occasional attacks of diarrhoea, mild pyrexia and enlarged liver. The other cases showed symptoms of advanced stages of the disease, namely, jaundice, ascites and attacks of pyrexia. The age of the patients ranged from 1½ years to 4 years. Two patients were females and two were males. Two out of the four cases showed both fatty infiltration and co-existent areas of focal necrosis. The remaining two cases showed marked fibrosis but neither the pre-existent fatty infiltration nor focal necrosis.

Table 2 summarises the broad clinical features of these cases along with the histological finding in liver biopsy.

TABLE 2—SHOWING CLINICAL FEATURES AND HISTOLOGY IN INFANTILE CIRRHOSIS

Age in in years /Sex	Clinical features	Histopathological findings
1¼/m	Liver enlarged—duration 6 months; severe jaundice—15 days.	Pericellular type of fibrosis, extensive infiltration with polymorphs. No fatty degeneration. No necrosis.
1½/m	Fever off and on—duration 6 months; enlarged liver—6 months; jaundice.	Marked fibrous tissue hyperplasia in a hyperplastic liver nodule (no necrosis, no fatty degeneration).
1½/f	Fever with diarrhoea, anaemia, liver enlarged one finger.	Pericellular types of fibrosis, fatty degeneration, slight focal necrosis, round cell infiltration.
4/f	Ascites—3 months; diarrhoea and pyrexia—1 year.	Lobular and lamellar pattern lost, irregularly disposed, thin strands of fibrous tissue, marked fatty degeneration, slight focal necrosis and marked reticulo-endothelial hyperplasia.

FOCAL NECROSIS OF THE LIVER

Focal necrosis was the commonest lesion encountered in our work. One hundred and thirty five cases out of a total of 76 autopsies and 331 liver biopsy specimens showed focal necrosis along with mononuclear infiltration with or without reticulo-endothelial cell hyperplasia—a lesion sometimes referred to as 'subacute hepatitis'. Thirty five cases showed central necrosis. One case of diffuse necrosis was encountered. No case of pure mid-zonal or peripheral necrosis was seen.

Table 3 shows the diseases associated with focal necrosis (331 biopsies and 76 autopsies)

The following interesting observations were made:

Focal necrosis in pulmonary tuberculosis was not accompanied by tuberculous granuloma except in 2 cases. Six serial sections were examined in each case.

In none of the liver lesions in amoebiasis, *E. histolytica* was seen. The lesions of the liver in this disease could not be differentiated from those seen in tuberculous enterocolitis and diarrhoea cases.

TABLE 3—SHOWING CLINICAL FEATURES IN FOCAL NECROSIS

Clinical conditions	No. of cases examined	No. of cases showing focal necrosis
Pulmonary tuberculosis ...	34	26
Tuberculous ileocolitis ...	8	6
Amoebiasis :		
With liver enlarged ...	28	25
Liver not palpable ...	3	2
Anaemia (of various types) ...	25	17
Diarrhoea	1	1
Infectious hepatitis (acute) ...	6	3
Infectious hepatitis (subacute) ...	2	2
Splenomegaly and hepatomegaly	10	5
Cardiac failure	10	2
Nephritis or nephrosis or pyelonephritis	5	5
Kwashiorkor	8	2
Cholecystitis	1	1
Diabetes mellitus	4	2
Early cirrhotic liver	5	2
Cirrhosis liver	40	24
Infantile cirrhosis	8	3
Cancer liver	6	4
Tuberculosis (generalised) ...	1	1
Malaria	2	2

A high incidence (17 cases out of 25 biopsies examined) of necrosis in anaemia cases was noteworthy. The remaining cases showed either normal livers or fatty infiltration. These cases of anaemia were due mostly to either hookworm infestation or were of nutritional macrocytic type.

The results of the study of cases showing central necrosis are summarised in Table 4.

TABLE 4—SHOWING INCIDENCE OF CENTRAL NECROSIS IN 407 CASES (331 BIOPSIES AND 76 AUTOPSIES)

Disease	No. of cases showing necrosis
Chronic venous congestion	10
Acute cardiac failure or shock ...	10
Tuberculosis	1
Infective hepatitis	3

N.B.—Cases showing postmortem autolysis have not been taken into account.

All the cases of shock were from medicolegal autopsies, i.e., persons who had met with a sudden death.

A further analysis of lesions in the liver encountered in pulmonary tuberculosis, anaemia and tuberculous abdomen showed that these lesions could not be distinguished from one another. All of them showed focal necrosis, mononuclear infiltration in periportal areas and associated with or without reticuloendothelial hyperplasia.

DISCUSSION

Our findings in cirrhosis of the liver cases are interesting. Twenty-one cases out of 40 showed besides irregularly disposed bands of fibrous tissue, evidence of focal necrosis but no fatty infiltration. Because of the relatively diffuse bands of fibrous tissue and mononuclear infiltration in these cases, we have interpreted them as cirrhosis of postnecrotic variety. The remaining 14 cases showed fine strands of fibrous tissue, irregularly disposed collagen fibres along with fatty infiltration and were, therefore, diagnosed as cirrhosis of 'diffuse hepatic fibrosis' variety (Himmsworth, 1947). A number of reports have appeared in the literature giving relative incidence of these two types of cirrhosis. Dible (1951) reporting from England found that 39 cases out of the 69 autopsies in cases of cirrhosis of the liver he examined were not preceded by fatty infiltration and were of the postnecrotic variety. The main consideration which led him to make this diagnosis was the absence of fatty infiltration in the liver in these cases. Veliath and Srinivasachar (1951) from Southern India reported that 52·9 per cent of the cases of cirrhosis of the liver seen by them were of the 'postnecrotic variety', and the remaining cases were of the 'diffuse hepatic fibrosis' variety, indicating that postnecrotic type of cirrhosis is not uncommon in this country. Our figures for various types of cirrhosis, as already indicated are 52·5 per cent cases of postnecrotic variety, 35 per cent of diffuse hepatic fibrosis variety, 7·5 per cent of zeoparasitic and 5 per cent of cardiac cirrhosis varieties (5 per cent of cases showed both focal necrosis and fatty degeneration).

A number of workers have recently claimed that dietary deficiency *per se* is unable to lead to cirrhosis of liver (Walters and Waterlow, 1954; Lie and Sutomo *et al*, 1955). They insist that additional factors, may be infections or toxaemia, operate in addition to dietary deficiency before a condition of cirrhosis sets in. Evidence of malarial infection in three of our cases of cirrhosis liver is interesting. In this regard it may be further stated that some workers have found focal necrosis associated with diseases of a very varied nature and in their opinion food deficiency coupled with any strong irritation will lead to focal necrosis.

And repeated attacks of focal necrosis are known experimentally to lead to cirrhosis liver (Himmsworth, 1947).

Necrosis of the liver is attributed to various causes. These causes include chemical poisons, vegetable poisons, various types of toxaemia and infections and dietetic deficiencies. It is interesting to note that authors like Spellberg (1954) and Litchman (1949) do not mention anaemia as a cause of necrosis. Spellberg (1954) describing liver lesions in anaemia, mentions only fatty degeneration and adds that the liver is more susceptible to circulating toxins. Standard works on haematology (Wintrobe, 1951; Whitby and Britton, 1953) are silent about lesions in various types of anaemias (except anaemias associated with splenomegaly). Consequently findings of focal necrosis in our anaemia cases are interesting. 17 cases out of 25 showed focal necrosis, while only 8 showed the classical lesions of fatty infiltration. The discrepancy between the classical descriptions and the lesions encountered in our cases shows the possibility that an aetiological factor, in addition to anaemia, is at work in causing the necrotic lesions. A surmise with regard to this additional cause can be made from a consideration of the aetiology of hookworm anaemia. Hookworm anaemia is known to develop only in those persons in whom hookworm infestation is associated with deficient protein in diet, (Napier, 1946; Whitby and Britton, 1953; Delangen, 1933). Further Stewart (1933) has shown that hookworm infections lessen protein digestion. The protein deficiency thus caused by hookworm infestation and the deficiency in diet could easily explain the liver necrosis encountered in our cases of hookworm anaemia. It is easy to extend this explanation for the liver lesions encountered in nutritional macrocytic anaemia cases.

Study of the lesions in amoebiasis, diarrhoea and tuberculous abdomen were identical in all the three disorders mentioned and also indistinguishable from the necrotic lesions encountered in our anaemia cases. Since the liver lesions in amoebiasis were not associated with the presence of *E. histolytica* at the site of the lesions, and the lesions in tuberculous abdomen were not associated with the typical granuloma, the non-specific nature of these lesions is evident. All these three gastro-intestinal disorders are likely to be associated with poor digestion or/and poor absorption of food as a result of diarrhoea. Could not then the poor food absorption explain these lesions? However, all these cases were not suffering from diarrhoea, the question naturally arose 'Is the diet of the populace we are working with deficient in factors like cystine, the deficiency of which causes necrosis?'

In this regard presence of focal necrosis in the majority of our cases of pulmonary tuberculosis in contrast to the classical lesion of fatty infiltration (Spellberg, 1954) in the liver in this disease is interesting as pointing to the same conclusion as stated above.

The above observations lead us to the study of the diet of the people we are working with. Most of the patients from whom the above material was collected belong to the labouring classes working on the agricultural fields. The cereals used by this group of people in different parts of Rajasthan, are *bajra* in Jodhpur and Bikaner (wheat being used only by a few), while in Jaipur barley and gram are consumed in summer and *bajra* in winter. In Udaipur the staple diet is maize. Green vegetables are scarcely taken. Curd and milk are woefully lacking, though whey may be taken sometimes. It is evident that diet is poor in animal proteins. A study of the protein and amino acid content of cereal maize shows that while whole maize is fairly rich in protein (11·1 per cent), in the maize flour the protein content is quite poor (0·6 per cent) (Aykroyd, 1941). Further maize is poorer in gluten than wheat and the cystine content of maize protein is only 1·5 per cent as compared with the cystine content of wheat which is 1·8 per cent and of eggs which stands at 2·4 per cent. A deficiency in this amino acid is thus evident in the diet of the maize-consuming populace of Udaipur. Deficiency in cystine is known to predispose the liver to necrosis (Himmsworth, 1947) by various non-specific irritants. This deficiency is likely to be responsible for the lesions seen in our cases. Corresponding figures for cystine content of bajra and barley proteins are not available. It is possible that their cystine content is low. Elucidation of the truth of the statement requires a probe into the problem. Herein is thus a field for the specialist in nutrition to provide the necessary answer.

Summary

An analysis of the results of histopathological examination of the livers from 331 biopsies and 76 autopsies was made with particular reference to certain lesions of the liver.

A high incidence of postnecrotic type of cirrhosis as contrasted with diffuse hepatic fibrosis was encountered in the series under review.

Focal necrosis was the commonest lesion encountered—the various diseases with which it was found associated are given in detail.

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2-BENZYL-IMIDAZOLINE HYDROCHLORIDE IN HEMIPLEGIA

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A synthetic sympatholytic drug with the chemical structure 2-benzyl-4-5-imidazoline hydrochloride has been used with some success in cases of hemiplegia. It has in its structure a histamine-like component as well as a component which is present in adrenaline. It appears that the drug exerts its action by competing with adrenergic substances reversing their action. It has a direct dilating effect on the walls of the vessels, and acts as a sympatholytic drug blocking the sympathetic nerves at their termination in the vascular musculature, thus preventing the transmission of the vasoconstricting excitatory stimuli. It also has an anti-adrenergic effect blocking the vasoconstricting action of circulating adrenaline or noradrenaline. This drug has been used extensively in the treatment of peripheral vascular disorders.

The rationale of the therapy is the same as that of stellate ganglion block. In cerebrovascular conditions, there is always an associated reflex spasm of the vessel due to sympathetic stimulation. This results in widespread disturbances in the functions

of the body. The vasodilating effect relieves the vascular spasm.

Intra-arterial injection of 0·2 to 0·5 c.c. of a 25 mg. per c.c. solution, into the ipsilateral carotid artery in acute cerebrovascular disorders have been used, and remarkable results especially in cases of aphasia have been achieved.

Although reports point to the use of the drug in the acute stage of cerebrovascular disorders, its use in the chronic stages of the disease is also helpful, if there are no permanent deformities as contractures and pronounced disuse wasting. The drug exerts an ameliorating effect even in the late stage of the disease.

MATERIAL

Fifty cases were treated in Stanley Hospital, Madras. All the patients were males and the thrombosis was of acute onset. The age varied from 20-68 years.

20-30 years	.. ..	10 cases
31-40 ,,	.. ..	12 ,,
41-50 ,,	.. ..	19 ,,
Over 50 years	.. ..	9 ,,
	Total ..	50 cases

Of the fifty cases, thrombosis was of syphilitic origin in 14 cases, hypertensive in 27 cases and arteriosclerotic in origin in 9 cases. Eighteen of the 50 cases were diabetic.

In four cases the thrombosis was in the Heubner's artery, a branch of the anterior cerebral artery with paralysis involving the face and upper extremity—the lower extremity was unaffected. In 36 cases the thrombosis occurred in the right internal capsule (left-sided hemiplegia). On this series of 36 cases speech was affected in three right-handed individuals. Thrombosis affected the left internal capsule in 14 cases in 10 of whom speech was involved. In none of the 50 cases was hemianaesthesia noted.

Weeks	No. of cases
0- 1	.. 14
2- 4	.. 20
5-12	.. 6
13-52	.. 4

DOSAGE

10 mg. of 2-benzyl-imidazoline hydrochloride (1 amp.) was given intravenously twice daily till there was appreciable improvement—on an average this took three days. Later, 1 amp. was administered daily. The patient was usually able to walk after about the 5th or 6th injection,

abduct the arm fully after the 10th and dorsiflex his hands after the 15th. Injections were continued upto a total of 300 mg. Oral medication was then instituted, the patients were given one tablet three times a day. The patients were asked to take 1 tablet t.d.s. for the rest of their lives.

Patients were hospitalised only for a week and were discharged with the onset of the ambulatory stage with advice to continue the drug for the rest of their lives.

Results

The response was good in inflammatory conditions, syphilis, and in thrombosis occurring in young hypertensives. In arteriosclerosis the response was slow, but gratifying.

Thrombosis was avoided in the 6 cases seen in the prodromal stage. Complete recovery within a week was noted in all the 6 cases. Complete recovery with slight weakness of the affected side and loss of flexion of the foot was observed in the 14 cases seen within a week. This result was achieved at the end of 8-12 weeks. Good relief and functional recovery occurred in the 20 cases seen within 4 weeks of onset. Patients were asked to walk with ease (without the usual hemiplegic gait), use their arms freely for their work, i.e., lifting weights, tying knots, carrying things, rubbing the body, etc. Wasting of limbs was absent. A slight degree of paresis persisted. In the remaining cases the ability to walk with ease was restored with some functional recovery in the upper extremity. Finer movements of the hand were carried out with some difficulty. Deformity and wasting were not seen.

Normal speech was restored in 8 cases. A certain amount of dysarthria persisted in the remaining cases. In longstanding cases of hemiplegia the response was very slow. The wasting of muscles seen in cases prior to treatment were unaffected, by the treatment. Some cases under treatment showed occasional sudden nocturnal jerks of the lower limbs. During the early part of the treatment, emotional imbalance, personality changes and loss of memory were noted.

There was a fall of blood pressure both systolic and diastolic especially in cases of hypertension treated with intramuscular injections of the drug. Excessive sweating on the hemiplegic side, oedema of the affected side, and sialorrhoea were noted after intravenous injections in some cases. Such findings were more common in hypertensives and did not occur in arteriosclerotics.

Hiccough was noted in four cases after i.v. injections and was controlled by atropine sulphate injections and magnesium trisilicate (one teaspoonful thrice daily) by mouth.

Follow-up after discharge from hospital—Beneficial results were maintained in those who continued to take the drug regularly. The disabilities are gradually becoming less and less. The blood pressure gradually mounted up. The blood pressure was below the initial blood pressure prior to the cerebral attack and above the blood pressure during the attack. In one patient prior to the hemiplegia B.P. was 240/140 mm. of Hg. During the attack it was 150-170/90-105. After treatment B.P. rose upto 170/110. As the patient showed functional recovery, B.P. rose up. The patients who completely recovered had always higher blood pressure than normal blood pressure for their age.

In those patients who discontinued treatment after a month or two after discharge from the hospital, further improvement was very slight. In 2 cases where treatment was discontinued after 2 months the patients developed hemiplegia one on the same side and the other on the opposite side. The second attack in each case developed after 6 months following the first attack. The response to treatment of the second attack was not so favourable as that of the first one. It was very slow and the recovery was not very appreciable.

Toxicity and Side Effects

Excessive salivation, profuse sweating (a good sign of vasodilatation) on the paralysed side, transient oedema on the paralysed side, gastric irritation and hiccough were noted. With the exception of hiccough the other symptoms did not contra-indicate the continuance of the drug. Hiccough is often noted in elderly arteriosclerotics. The onset of hiccough should be an indication for the stoppage of 2-benzyl-imidazoline hydrochloride and the administration of atropine (1/100 gr. subcutaneously).

Summary

The mode of action of 2-benzyl-imidazoline hydrochloride, its dosage and toxic effects and other side-reactions are mentioned.

Results of treatment of fifty cases treated with this drug have been discussed.

Acknowledgment

My thanks are due to the Dean, Government Stanley Hospital for permission to use the hospital records. I also acknowledge with thanks the help and guidance given by Drs. G. Victor and Ananthachari under whom the cases were admitted and treated and to all the house physicians who helped and co-operated in carrying out treatment and in maintaining records. I render my grateful thanks especially to Drs. Janaki, T. C. Saraswathi, Swaminathan and Madhavan Kutty.

PRACTITIONERS' SERIES

INFECTIVE HEPATITIS

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Inflammatory affections of the liver caused by viruses have been in the forefront of pathological, bacteriological and clinical interest since large outbreaks of epidemic jaundice occurred during the last war. They were of two kinds. The one, afflicting many thousands of people, especially in Italy and the Middle East, where troops or prisoners lived under primitive camp conditions, were due to infective or infectious or epidemic hepatitis, an enteral, probably food and water-borne infection, which we older clinicians knew previously as catarrhal jaundice. The other type was strictly man-made, acquired parenterally by vaccination, inoculation, injection, transfusion etc.; 0·0005 ml. blood is sufficient to transmit the infection. The largest outbreak occurred in the U.S. Army following mass vaccination against yellow fever and involved more than 50,000 men. Infective hepatitis and homologous serum hepatitis are caused by viruses called I.H. and S.H. respectively, sufficiently different from each other to cause two different diseases for all practical purposes. I.H. is found always in the faeces of the patient and at certain periods in his blood. S.H. occurs in the blood only. The incubation period of the former is two to six weeks, of the latter 2 to 5 months and each of these two diseases confers increased resistance only to its own virus; IH does not cause cross-immunity against S.H. or *vice versa*. Less definite are differences in age incidence, although IH occurs more frequently below age 30, SH above 20. IH shows more of a seasonal incidence similar to typhoid fever and poliomyelitis and its onset is often more dramatic with rigor and fever. SH might occur at any time of the year and starts insidiously with hardly any fever.

Pathology

Infective hepatitis presented an occasion for a classical error. Virchow, the father of cellular pathology, at the post-mortem of a man with catarrhal jaundice, killed in an accident, found a gastroduodenitis with a mucous plug, apparently occluding the papilla Vateri ; he concluded that every case of catarrhal jaundice is caused by the same mechanism of mucous obstruction (1865). Some 60 years later, Eppinger taught that even the mildest case of icterus catarrhalis is caused by hepatitis, presenting in miniature the changes leading to acute yellow atrophy which in fact might develop out of every case of catarrhal jaundice. The pathological findings of the last 10 years, based largely on aspiration liver biopsy, confirmed this theory. Acute viral hepatitis of both types, IH as well as SH, is characterised by centrilobular necrosis with periportal cellular infiltration and endothelial hyperplasia of the sinusoids. Surviving liver cells retain their normal glycogen contents and fatty changes are absent. An important feature for future regeneration is the preservation of the reticular framework. Bile thrombi and bile duct proliferation are prominent in surgical obstructive jaundice, not in viral hepatitis. Fatal cases show the fully developed picture of acute yellow atrophy if the course has been of the fulminating type, whereas in the liver of those who succumb after more than two weeks, yellow patches of necrosis alternate with red areas of haemorrhage and islands of regeneration.

Clinical Picture

The clinical picture of infective hepatitis differs from that of the Western authors in so far as the more dramatic features which in the West occur in a minority of cases are here almost the rule. Particularly at the beginning of epidemics most of the cases start exactly like an attack of acute malaria with bilious features. Rigor with fever reaching 102° or 103°, severe pain in the upper abdomen or around the umbilicus and persistent vomiting of every kind of food and drink terminate the picture. If antimalarials have been given at the onset, one does not know whether the very troublesome vomiting is due to the medication. Frequently, fever and abdominal distress subside after about 2 or 3 days, confirming apparently the diagnosis of malaria, only to be followed after another 3 to 4 days of comparative well-being by the appearance of dark urine and soon afterwards an icteric tinge of the sclerae. In other somewhat rarer cases, fever, abdominal pain and vomiting persist with varying intensity, increasing often during the night, for almost one week until bile pigments in the urine, and a day or two later in the sclerae, clear the diagnosis. The last group behaves as catarrhal jaundice used to do. A real or imagined dietary indiscretion is followed by one week of extreme anorexia ; there are few other conditions of such a strong aversion against food when even thinking of it causes discomfort or nausea. By the end of the week jaundice becomes manifest.

It is usually stated that with the appearance of jaundice, except in fulminating hepatitis, com-

plaints practically disappear, provided the patient takes rest and a reasonable diet. This is just as wrong as to believe that the patient ceases to be infectious within a few days of his becoming jaundiced. The cases are not rare where during the icteric stage the temperature upto 100°F continues and bouts of pain, nausea and even vomiting recur. However, milder infections form the majority ; when treated properly, they take a quieter course, although it is impossible to predict the outcome unless the first 8 or 10 days of declared jaundice are over, as fulminating cases usually become manifest by the end of the first week. In an otherwise healthy individual below the age of about 40, the acute stage lasts 3 to 4 weeks but even 6 to 8 weeks are no rarity ; by this time the clinical signs of jaundice, the liver enlargement with or without tenderness, and bilirubin in the urine should have disappeared and sedimentation rate and liver function tests returned to normal.

Quite different is the course in fulminating hepatitis which quickly leads to hepatocellular failure. Hepatic failure has no typical histological picture and, therefore, cannot be diagnosed by the histopathologist. The diagnosis has to be made by the clinician. The first warning signs of threatening failure are neurological changes and foetor hepaticus ; the latter gives the breath a sweetish and slightly faecal smell ; in acute failure it often precedes hepatic coma. It is probably due to nitrogenous metabolites which are detoxified by the normal liver but pass from the portal into the systemic circulation, once hepatic failure is imminent. The signals given off by the C.N.S. are still more ominous. In a case of infective hepatitis once the speech becomes slightly slurred, the general behaviour in any way unusual (mostly showing less self-control) or the patient's orientation faulty, hepatic coma may be expected to develop within hours.

As soon as coma has set in, it is not possible to rouse the patient even so far as to open or close his eyes or to show the tongue on request ; and yet, defensive reflexes are extremly strong, so that the patient may put up a vigorous fight against an intravenous drip. We find it often necessary, quite paradoxically, to give such a patient a paraldehyde enema before the needle for the glucose drip could be inserted. The patient lies there often with knees flexed to the abdomen and crossed legs, pupils dilated and not responding to light, eyes wandering, tendon jerks exaggerated and an extensor plantar reflex ; not rarely also an extrapyramidal syndrome can be observed consisting of so-called hepatic tremor and cog-wheel rigidity of striatal origin. Finally, Cheyne-Stokes' breathing and hyperthermia mark the fast approaching end.

Treatment

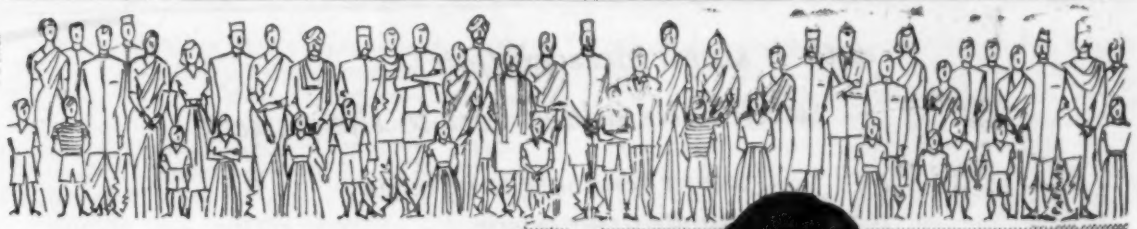
Prevention is difficult because the virus has not been isolated so far and experimental inoculation of animals has met with doubtful success ; although some workers reported to have produced hepatitis, nobody succeeded in retransmitting the infection to human volunteers who played such an important part in the elucidation of many problems of viral hepatitis. The virus is also extremely resistant to ordinary methods of water sterilisation so that nothing short of boiling for 10 minutes will destroy it.

On the other hand, gamma-globulin proved particularly effective in preventing infective, not serum hepatitis. In epidemic outbreaks in schools, camps, prisons etc., 0·02 ml. per kg. of body weight conferred a fairly long lasting immunity if given after exposure and upto 6 days before the expected onset of clinical disease. The protection seems to be due to passive-active immunisation.

Homologous serum hepatitis can be prevented to a large extent by painstaking sterilisation of every instrument, used for penetrating the skin or communicating with the blood stream : needles lancets, syringes, tubing used for connections must be boiled in water for at least 10 minutes and carefully washed immediately after use so as to prevent the drying of virus containing particles on surfaces. Inspite of all possible care, blood donors remain a potential source of infection as long as the virus cannot be identified in symptomless carriers ; to block this way of virus dissemination as far as feasible every previous jaundice patient and every one who was in contact with jaundice patients within the last year should be excluded as a blood donor ; how necessary such measures are, is shown by the fact that about one in 200 American blood donors is a carrier and that the virus retains its activity for a long time if the plasma is dried or frozen.

Carefully conducted follow-up studies of large numbers of both IH and SH patients, carried out for years, showed beyond doubt that cirrhosis hardly ever develops as a consequence of hepatitis.

Treatment is the same for both kinds of viral hepatitis. The one (till quite recently) undisputed factor, decisive for the quickest possible recovery is rest. Neither diet nor drugs are as important as the avoidance of physical activity as long as jaundice is present. Nobody should be allowed to get up until the icterus index is below 20, serum bilirubin below 2 mg. per cent, cephalin-cholesterol flocculation not more than 2 plus, and



off to a good start...

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thymol turbidity at the most 5 units. Quite recently a strongly dissenting voice was raised. An almost ideally controlled study was carried out by 8 selected doctors on behalf of the Hepatitis Committee of the Commission on Liver Diseases of the U.S. Armed Forces Epidemiological Board on 460 male members of the U.S. Armed Forces suffering from infective hepatitis ; they all came from Korea or Japan and were treated at the Hepatitis Centre in Kyoto. To cut a 70 page report short, the result was that a patient should be forced to stay in bed only as long as he felt sick ; as soon as symptoms have subsided, irrespective of the degree of jaundice etc., he was encouraged to move freely in the ward with compulsory rest of 1 hour after meals only. The regime along with a very high protein high fat diet, reduced the duration of illness by about one week compared with cases treated on conservative lines. The authors try to explain the difference between their results and practically all preceding ones, including those obtained by other workers in the U.S. Army ; apart from some differences in the character of the disease as observed in Italy in 1945, it is assumed that the nutritional status of the patients in the present study was perhaps better than that during some World War II epidemics. The latter point would make it abundantly clear that results achieved with American soldiers cannot be applied to our patients because a protein starved liver will react quite differently from one saturated with first class proteins.

Diet has been a subject of controversy for years. The excellent results of high protein diet observed by Patek and Post in Laennec's cirrhosis have been straight away introduced into the treatment of infective hepatitis, until recently Sheila Shercock warned against the flooding with nitrogenous metabolites of a liver which might break down any time and which even before this critical moment might be unable to prevent the entry of such toxic products into the systemic circulation. However, the same mass investigation of 460 American soldiers led to the recommendation of a diet containing 150 g. protein and 150 g. fat, both figures extremely high for any but American digestive apparatuses. Before such dietary principles could be considered here, one would have to compare the diet to which the healthy American soldier has been accustomed with that of our hepatitis cases prior to their illness. In any case, the American investigation has made it very probable that the extreme starvation preferred by many doctors here is not justified. On the contrary, a liberal supply of milk fortified with skim milk powder or even, perhaps, with full milk powder is certainly beneficial whenever the patient can digest it. Carbohydrates in non-irritative form, pearl sago, boiled rice, finely ground wheat dalia, biscuits, honey, glucose or sugarcane juice may be given freely. Regarding fat, I am sure that fried, rich or greasy food must be avoided, while toast with butter or baked potato with *dahi*, a little cream added to a wheat gruel or stewed fruit might stimulate the appetite and so benefit the patient whose gastric function is paralysed almost as much by the monotonous diet as by the illness itself.

Quite different has to be the dietary regime in fulminating cases with threatening coma. Intravenous glucose with vitamin B-complex and vitamin C should be started at once and given at the rate of at least 1,000 c.c. of a 5 per cent solution per day. Fruit juice and a small amount of skim milk or "chach" should be given at short intervals if nausea or vomiting do not interfere with oral feeds.

Treatment of hepatic coma, which till recently seemed rather hopeless, nowadays yields the most gratifying results in medical practice. This change is due to the effect of the tetracyclines, used either alone or combined with cortisone and corticotropine.

The treatment recommended consists of tetracycline, 100 mg. twice a day i.v. or i.m., hydrocortisone 60-80 mg. a day and corticotropine, 30-40 mg. a day i.m. combined with a continuous glucose drip containing also vitamin B-complex and C, but without saline in the presence of cortisone.

Based on a fairly extensive experience—hepatitis cases treated with chlor- or oxy-tetracycline going back to 1950—it is firmly believed that a condition with such a tremendously high mortality as hepatic coma in fulminating hepatitis requires the best possible therapeutic help and this is provided, in our opinion, only by the combination of tetracyclines with the cortisone-corticotropine group.

LIVER FUNCTION TESTS †

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In the liver lobule there are three separate units from the functional point of view : (i) parenchymatous cells for hepatocellular functions which

† Paper read at XXXII All-India Medical Conference in the Symposium on Liver Diseases, Jaipur, 1955.

TABLE 1—FUNCTIONS OF THE LIVER AS BASIS OF TESTS

1. Metabolic functions ...	Carbohydrates		Glucose Fructose Galactose ...	...	Galactose tolerance test oral or intravenous
	Proteins and aminoacids ...		...	...	Serum albumin and globulin
	Fats and sterols ...		...	...	Total and esterified cholesterol
	Vitamins ...	...	A and K ...	...	Prothrombin
	Water				
2. Haematological functions ...	Haemopoiesis Coagulation		Prothrombin ...	...	Prothrombin response to K intravenously
3. Detoxicating conjugation of x with y to form z	x	y	z		
	Sulphuric acid	Indoxyl	Indican		
	Glucuronic acid	Phenols	Glucuronides		
	Acetic acid	Aminesamides	Acetyl sulphonamides		
	Glycine	Benzoic acid	Hippuric acid		Hippuric acid
4. Bile formation	Bilirubin excretion		... ...	...	Serum bilirubin
	Urobilinogen excretion		... ...	...	Urine bilirubin Urine urobilinogen Faeces urobilinogen
5. Enzymes manufacture ...	Prothrombin Acetyl cholinesterase Phosphatase Amylase				Serum alkaline phosphatase
6. Excretory function ...	Excretion of dyes ...		... ...	...	Bromsulphthalein retention

are mostly metabolic, (ii) beginnings of the biliary tract for cholangiolar type of function, and (iii) Kupffer cells for function of the reticulo-endothelial cells. The functions of the liver as basis of various tests are detailed in Table 1.

Before the consideration of individual tests one must realise that there are limitations in their interpretations because of the multiplicity and complexity of the functions of this organ. With one anatomic unit there are three functional units, hepatocellular, cholangiolar, and reticulo-endothelial. It would be an unusual pathological process that would affect all these functions to the same extent. All the functions of the liver proceed independent of one another, and marked impairment in one function may very well go along with normal function in another. Also functional tests may give varying results from day to day and some of the tests should be done serially. The liver possesses a remarkable power of regeneration. Clinically simultaneous regenerative processes may compensate and completely mask the damage. Apart from the regenerative power there is a great reserve power or margin of safety that exists in the liver. Eighty per cent of the liver tissues should be damaged before abnormal results are obtained. In liver disease one test is useless and combination of tests is more useful to get an integrated and balanced picture of the liver function, particularly for early and mild disturbances.

Liver function tests are a valuable supplement to but no substitute for a well-taken history and a thorough physical examination. The interpretation of the same depends on the understanding of the physiological principles, sources of error, skill and reliability of the persons collecting the samples and performing the tests. The results at times may be misleading in respect to diagnosis unless interpreted in relation to the history, physical examination, stage and course of the disease, and the results of other diagnostic procedures, as for example,

1. In hepatic disease with little damage to the polygonal cells as in arsenical hepatitis, and virus hepatitis, the picture of extrahepatic obstruction may be obtained.
2. In infectious diseases like malaria, infectious mononucleosis, Weil's disease, yellow fever, pneumonia, and septicaemia, changes in liver functions, which are not primarily liver diseases, may be obtained.
3. Neoplastic processes, benign tumours, cysts of the liver, may or may not show changes in hepatic function, and normal results do not exclude their presence.
4. Haemolytic jaundice with secondary liver involvement is common. Some tests are positive and haemolytic factor is discovered by thorough haematological investigations, including faeces urobilinogen studies.

5. Liver function may be impaired in severe thyrotoxicosis, and chronic passive congestion secondary to heart disease.

Liver function tests are not specific for any particular type of disease of the liver, and do not differentiate primary and secondary involvement of the liver. Some of the symptoms and signs which are the indications for various liver function tests are jaundice, enlarged liver, nodular liver, deep amber urine, foetor hepaticus, fatty stools, purpura and splenomegaly.

The various liver function tests can be divided into the following groups:

Group I—Material collected by practitioner, and of routine use.	1. Serum bilirubin 2. Serum alkaline phosphatase 3. Serum flocculation tests Colloidal gold Thymol turbidity and flocculation after 18 hours Cephaline cholesterol test 4. Serum albumin and globulin estimation
(*i*) 10 c.c. clotted blood free from haemolysis	
(*ii*) Urine preferably afternoon specimen	5. Urine bilirubin 6. Urine urobilinogen
Group II—More elaborate tests require attendance of the patient in the laboratory or a visit by the pathologist	1. Quantitative estimation of urine bile-pigments 2. Bromsulphthalein retention 3. Prothrombin response to vitamin K 4. Galactose tolerance test 5. Hippuric acid test
Group III—More simple tests not requiring special technical skill	1. Icteric index 2. Urine bilirubin by Harrison spot test and methyline blue test 3. Urine urobilinogen by Wallace and Diamond technique 4. Thymol test 5. Bromsulphthalein test

Group I tests are to be used as a routine and these are able to give the diagnosis of jaundice in the pre-icteric stage and to give the correct idea of its intensity and also to enable differentiation of the cause of jaundice. In a non-jaundiced patient these confirm the suggested hepatic disease. All the tests in group II are rarely required and often one or two are enough to give a correct diagnosis, as for example, for confirming liver cirrhosis serum albumin and globulin estimation and bromsulphthalein retention tests are the ones whihch are necessary. Prothrombin response to vitamin K is specifically indicated for prognosis in surgical cases of jaundice and to indicate surgical risk.

Table 2 gives the indications for liver function tests.

TABLE 2—SHOWING INDICATIONS FOR LIVER FUNCTION TESTS

SUSPECTED PRIMARY LIVER DISEASE	
Acute	
Infective hepatitis:	
Pre-icteric stage ...	Urine bilirubin +
Icteric stage ...	All pigmentary tests + Flocculation tests +
Chronic	
Hepatic cirrhosis ...	Pigmentary tests and other tests often – Serum protein estimation and bromsulphthalein retention tests useful.
SECONDARY INVOLVEMENT OF THE LIVER	
Heart failure	Turbidity and flocculation test + Urobilinuria +
Malaria Glandular fever Thyrotoxicosis	Flocculation tests + Galactose tolerance test Hippuric test +
ENLARGED LIVER	
Hepatic cirrhosis	Serum proteins Flocculation tests
Carcinoma	Raised serum alkaline phosphatase
JAUNDICE	
Hepatic cirrhosis ... ... Obstructive ... ... Haemolytic	Thymol flocculation Alkaline phosphatase } 80% Indirect serum bilirubin raised. Serum alkaline phosphatase normal.
PROGNOSTIC OF LIVER DISEASE	
Cirrhosis: Irrecoverable damage Poor prognosis	Serum albumin below 2%
Obstructive jaundice: Grave liver damage Poor operative risk	Prothrombin response to vitamin K intravenously absent
ANAEMIAS	
Haemolytic anaemia Pernicious anaemia	Serum bilirubin differential estimation. Indirect bilirubin raised.

The various tests may be now considered individually in relation to their usefulness in the diagnosis of liver diseases.

Serum bilirubin—The separate estimation of direct and indirect serum bilirubin by photo-electric method based on that of Malloy and Evelyn is possible. Direct pigment refers to colour developed in aqueous solution after 15 minutes, while the total pigment is read in alcoholic solution at 30 minutes. The indirect pigment represents the difference between the two values. The normal total serum bilirubin is from 0 to 1 mg. per 100 ml. of serum, latent jaundice exists between 1 and 3 mg. per 100 ml., patients with over 3 mg. are usually clinically jaundiced. Latent jaundice occurs in hepatic cirrhosis, infective hepatitis, and haemolytic anaemias, and demonstration of this by this determination is an important aid to diagnosis. In jaundiced patients both the direct and the indirect bilirubin are increased, but if more than half of the total is indirect the jaundice is haemolytic. When direct pigment is mainly increased the jaundice is either obstructive, or toxic and infective. This is also indicative accurately of the intensity of the jaundice. This may remain normal in chronic liver diseases, cirrhosis and carcinomatosis.

Serum alkaline phosphatase—The serum alkaline phosphatase is an enzyme produced mainly in the bones by the osteoblasts and also by the liver cells. This can only be used as liver function test when the bones are normal. Its level in the blood (normal 3 to 13 King-Armstrong units is raised in obstructive jaundice, hepatitis, carcinoma, abscess, amyloidosis, and cirrhosis. Values higher than 35 units are suggestive of obstruction in a jaundiced patient. In differential diagnosis of jaundice this test is combined with one of the flocculation tests.

Flocculation tests—Serum colloidal gold, reaction, thymol turbidity and flocculation test and cephalin-cholesterol flocculation test are used. The results vary individually. Thymol and gold tests are positive in 90 per cent cases of acute infective hepatitis, in 50 per cent cases of cirrhosis and negative in obstructive jaundice. Their value is in detection of hepatitis and in the differential diagnosis of jaundice. These are not specific tests of liver damage and are positive in glandular fever, and malaria and infective endocarditis due to serum protein disturbance. The clinical picture is especially to be taken into consideration in interpreting these tests. Normal sera give turbidity 0-4 units and no flocculation in 18 hours and 1+is definitely abnormal.

Serum albumin and globulin estimations—These are of most value in the more chronic type of liver disease as cirrhosis. Here albumin tends to fall and globulin rises. Normal albumin being 4 to 5·5 mg. per 100 ml. and globulin 1·5 to 3·0 g. per 100 ml. A/G ratio thus falls. These confirm the diagnosis of cirrhosis. Albumin below 2 per cent indicates poor prognosis and often associated with oedema. High globulin does not produce symptoms.

Qualitative urine tests for bilirubin and urobilinogen—The foam and iodine tests for bile are insensitive. For bilirubin, Harrison Fouchet test and for urobilinogen Ehrlich-Watson test are useful. In the absence of clinical jaundice the presence of bilirubin in the urine indicates latent jaundice as in hepatitis or biliary obstruction and the test is particularly valuable in the pre-icteric stage of hepatitis and in hepatic cirrhosis. Increase of urine urobilinogen may occur in latent jaundice, and in chronic liver damage of hepatic cirrhosis. It is less specific indication of liver pathology than bilirubin in urine. This may occur temporarily in fevers, increase is suggestive of toxic and infective jaundice. Normally in 1 : 10 diluted urine it is negative but sometimes 1 : 20 and 1 : 30 it may be positive and so 1 : 30 and more dilution is taken positive.

Quantitative estimation of bile pigments—This test is particularly indicated in non-jaundiced patients as cases of hepatitis and cirrhosis. This is more accurate and reliable than the qualitative test. One or two hour specimen of the afternoon is used for estimation of this pigment, and normal values are 0 to 1 Ehrlich unit per 2 hours or upto 3 mg. per 24 hours. Urinary excretion is increased as a result of liver damage.

Prothrombin response to vitamin K—This is used mainly in cases of obstructive jaundice but is also of value for differentiation of surgical and non-surgical jaundice. It is applicable only to those patients who have a low prothrombin value when first seen. Low prothrombin is either due to lack of absorption from the intestine in cases of obstructive jaundice due to lack of bile from the intestine or due to severe liver damage as in cases of hepatitis and cirrhosis. About 25 per cent of all jaundiced patients show the initial low prothrombin level. After the initial estimation water soluble preparation of vitamin K is given I.V. and prothrombin is again estimated by modified Quick's technique after 18 hours of the injection. A rapid return to normal indicates good liver function and confirms biliary obstruction. Absence of response indicates gross damage and suggests the hepatogenous origin of jaundice. A low prothrombin after vitamin K therapy is significant. The test is of great importance if opera-

tion is contemplated and a low level indicates greatly increased risk of haemorrhage.

Bromsulphthalein retention—It is a harmless dye which is injected intravenously and is excreted mainly in the urine. 5 mg. per kg. body weight is injected and blood is collected exactly 45 minutes after the injection. The amount of dye remaining in the serum is estimated. Normal persons show no significant retention and in pathological states the amount remaining in the circulation is expressed as percentage retention. It is of not much value in jaundiced patients and is mainly used in non-jaundiced patients as cases of hepatitis and cirrhosis. It is a sensitive test and even this is not always positive in cirrhosis. It also indicates liver dysfunction in conditions like malaria, diabetes mellitus, and glandular fever.

Galactose tolerance test—After giving 40 g. by mouth the urine is collected for five hours and the total amount normally should not be more than three grams and more than 3 g. is abnormal. Blood galactose level may be determined after oral administration or after intravenous injection. This is the most useful test for evaluating the course of severe, diffuse damage to hepatic cells.

Hippuric acid test—Quicks oral hippuric acid test is based on the assumption that the liver is the primary site of the synthesis of glycine, which it can combine with benzoic acid to form hippuric test. With an intake of 6 g. of benzoate by mouth a normal person excretes in the urine 3 g. of benzoic acid within 4 hours. Cases of hepatic dysfunction excrete less. This is the most useful test for evaluating the severity of moderate impaired function when renal failure is excluded.

Table 3 gives results of the tests useful in evaluating the functional status of the cells of the parenchyma in health and in patchy and diffuse disease of the liver. It clearly reveals the limitations which are due to tremendous reserve and the great regenerative capacity of the liver. Until this reserve and any functional regenerated cells are reduced to about 20 per cent of those in the normal liver, patchy lesions may show no evidence of impaired liver function and even the most extensive destruction by patchy lesions seldom shows more impairment than mild diffuse damage. These tests are arranged in the order of decreasing sensitivity. Tests in the lower half of Table 3 are positive in severe diffuse damage as in acute and sub-acute hepatic necrosis and so these are not of diagnostic value but good for prognosis, guiding treatment and evaluating the severity of damage in cases where the tests in the upper half are positive.

The more simple tests as mentioned in group III (*vide supra*) are such which do not require special technique and out of these the last three have already been dealt with and only the first two need be mentioned here. For icteric index the yellow colour of the serum is compared with that of the standard potassium bichromate solution. The normal range is 4 : 6 units. An index greater than 9 units usually indicates abnormally high concentration of serum bilirubin. Common sources of error are haemolysis lipaemia and carotenaemia and fasting specimen before breakfast should be used. Jaundice usually is not visible clinically until the icteric index reaches 15 or 20.

In Harrison's spot test for bilirubin the basis is the colour reaction on the addition of Fouchet's reagents to precipitate from urine with the addition of barium chloride and if bile pigment is present a blue, blue green or green colour results and the test may be graded as one positive to four positive depending whether weak blue or intense blue or blue green colour develops. A more simple test is the use of 0·2 per cent aqueous solution of methylene blue and the number of drops required to give a final blue colour to 5 c.c. of urine in a test tube is noted. The number of drops with which the green colour persists, i.e., one less than

TABLE 3—SHOWING TESTS USEFUL IN EVALUATING HEPATOCELLULAR DAMAGE

Useful test	Normal	Patchy	Diffuse mild
Bromsulphthalein 5 mg/kg. ...	5%	15%	15% retention
Thymol turbidity	0-4 units	5-10 units	11-35 units
Hippuric acid after 6 g. sodium benzoate	4·2-5·9 g./4 hrs.	3-5 g./4 hrs.	0-3 g./4 hrs.
Galactose tolerance after 40 g. oral	under 3 g./5 hrs.	2-5 g./5 hrs.	over 5 g./5 hrs.
Prothrombin time after vit. K 2 mg. I.V.	Normal	Improves	No improvement
Hypoglycaemia	Absent	Slight or absent	Pronounced
Tyrosinuria	0	0	do.

the number required to change the colour from green to blue is noted. Readings of five or more drops are almost always due to the presence of bilirubin in the urine and are taken as positive. Increasing readings from 5 drops to 10 or 15 indicate increasing jaundice and decreasing titration denotes decreasing jaundice. In early hepatitis the test is positive before clinical jaundice appears and before the total serum bilirubin is raised.

Summary

A liver function test alone can never be expected to establish a diagnosis.

Liver function tests indicate the presence of disease affecting every liver cell and patchy diseases when more than 80 per cent of the parenchyma is involved. They will detect sub-clinical jaundice and help in differentiating various types as obstructive, haematogenous and parenchymatous.

The modern liver function tests have reached a high degree of sensitivity and will rarely fail to demonstrate any significant liver damage, if present. The disturbances of function in various non-hepatic diseases complicate the interpretation and for that the whole clinical picture should always be kept in view.

The ideal diagnosis as basis for treatment should indicate specially the exact anatomical sites of each lesion, the nature, stage and extent of pathological process and the functional disturbances from this pathological process and this is possible by co-relating the history, physical science, and the results of the tests here considered and specific tests for determining the cause.

Many American laboratories apply a battery of about a dozen liver function tests. Our more moderately equipped institutions are not in a position to make such a liberal application of laboratory techniques which are often confusing and it is necessary therefore to decide which tests from the various possible ones will best serve the purpose of the clinical pathologist in attempting to assist the physician in his diagnosis and prognosis of liver disease. Decisions as to diagnosis, prognosis and treatment must always rest finally with the clinician but the laboratory can make a useful contribution to the study of jaundice.

For the differentiation of jaundice phosphatase, bilirubin and thymol flocculation test plus urinary and faecal pigments are most useful. For the assessment of increasing hepatic cell damage in acute hepatitis the bilirubin, albumin and globulin give a fair degree of co-relation with the liver histology as seen in biopsy specimen.

SPECIAL ARTICLE

A SHORT NOTE ON HEPATOLOGY FROM THE EARLIEST TIMES UP TO ABOUT 1900 A.D.

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AND

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Prehistoric, Assyrian and Oriental

The earliest anatomical models were clay models of sheep's liver used in instructions in Babylonian Temple school about 2,000 B.C. The model, now in the British Museum, is subdivided into squares and covered with cuneiform writing furnishing prophetic prognostications for the peculiarities noted on the various parts of the liver. From hepatoscopy the Babylonians learnt the structure of the liver. The right and left lobe, the caudate lobe, the quadrate lobe, the portal fissure, the cystic duct, the hepatic duct, the gall bladder and the fossa of the umbilical vein are all indicated. Technical names were given. Similar bronze models with Etruscan inscriptions were found at Pianza (fifth century, B.C.). In the ancient Hittite Empire sites of Asia Minor also models of sheep's liver have been discovered. The liver as the source of blood, was regarded as the seat of the soul and as God identified himself with the sacrificial animal, to inspect the liver was to see into the soul of the animal and the mind of God. In the Bible mention is made of the ligaments of the liver. Babylonian hepatoscopy was the popular form of fortune-telling. Liver diseases and eye diseases form the centric feature of Babylonian as of Arabic pathology. The three dimentional ancient clay models have been shown to be far superior to the five-lobed livers of *Tabulae anatomicae* of Vesalius.

The Babylonian Talmud (fifth century of our era) is a collection of tales and discussions containing interesting anatomical and physiological data including those on the liver. Diseases of the liver particularly jaundice appear to have been common. There were also Talmudic laws in respect of animals found diseased on slaughtering, e.g., caseous degeneration of the liver, etc.

In the medicine of ancient China (Neiching, 1,000 B.C.) the liver was regarded as the storer of blood containing the soul and as such it was

considered as the "father and general of the heart —the king and director of the body."

"The ancient Hindu medical writers (1,000 to 600 B.C.) recognised a close functional relationship between the liver and the spleen in the circulation of body fluids. Charaka mentions these organs as the root of the ducts that bear the blood. Both Charaka and Susruta maintained that it was in these organs that the *tejas* part of the circulating fluid (*rasa*) derived from the finer part (*sukshma bhaga*) of the chyme (*rasa*) of the small intestine driven thither through the heart by the *prana vayu* through a *dhamani* trunk became purified and coloured red by the bile through the agency of *ranjakagni* and emerged from there as blood to nourish and build all tissues, *dhatus*, of the body. Bile is identified by Susruta with the digestive fire which digested food. It is, however, apparent from the location of the point of entrance of this bile into the digestive tract in the part between the stomach (*amasaya*) and intestines (*pakasaya*) that it is the combined hepaticopancreatic secretion which it referred to" (Ramachandra Rao, 1940). Hepatic disease and pathology figure very little in ancient Hindu medical writings because of the humoral basis of the concept of disease of those times.

Graecoroman

The Hippocratic school (460-370 B.C.) viewed the liver and the spleen as sites of origin of two humours: (1) the warm and dry yellow bile, and (2) the cold and black bile, respectively. The liver was also looked upon as the producer of blood. In Hippocratic writings the cirrhosis of the liver is also mentioned. Democretus's (460-362 B.C.) system of "soul" had the effect of describing three different seats of the soul, the "soul atoms" in the liver subserving the functions of desire, lust, greed and other earthly passions. In Aristotelian physiology (384-322 B.C.) the spleen and the liver were coupled as one functional unit and considered as hot organs mainly with a digestive role, furnishing the heat to the contiguous organ—the stomach—where the concoction of food took place. To Herophilus (335-280 B.C.) known as 'father of anatomy', the liver was an extension of the digestive tract and a seat of nourishment, a description adopted by Galen. Erasistratus (310-250 B.C.) who originated the theory of 'plethora' attributed the ascites of cirrhosis to the conditions of the plethora of the liver. Aretaeus of Cappadocia (3rd to 2nd C. B.C.) designated the liver as source of the veins and described it as a solidified mass of blood with functions of production of blood and bile. Food was presumably brought by the portal vein from the stomach to the liver, from where it was carried as blood by the vena cava to the heart. Rufus of Ephesus (fl. 1 C. B.C.) was first to describe the liver as porcine (five-lobed), a blunder which was perpetrated upto Vesalius. Celcus (1st C.A.D.) prescribed laxatives in acute diseases of the liver manifested by vomiting and hiccups and incision in the case of an abscess. Galen (130-200 A.D.) considered the liver as a source of natural spirits (vital and animal) which flowed into the rest of the body. He supposed that the liver exercised more or less separate attractive, retentive, alterative, and expulsive powers corresponding to the extraction of nutritive elements from the gastrointestinal tract, retention of these elements, conversion into blood and waste product bile, and expulsion of the bile. The food which is absorbed from the alimentary canal is carried by the portal vein to the liver where it is converted into crude blood (natural spirits) endowed with nutritive properties and passed on through the inferior vena cava to the heart (*cf.* Hindu concept).

Dark Middle Ages

The Galenic doctrines held sway and the Byzantine civilisation added very little to these.

European Renaissance

The middle of the 15th century saw a revolt led by great seekers after truth like Antonio Bennivini, Alessandro Benedict, Paracelsus and others against Galenic doctrines, dogmas and textual sanctions. This led to the overthrow of humoral pathology and dawn of rational medicine. Study of disease became anatomical and the position of the liver sank to that of an excretor of waste product—bile. Paracelsus (1493-1541 A.D.) and Van Helmont (1577-1644) forestalled, though vaguely, the biochemical era by drawing attention to the chemical changes occurring in tissues and organs in health and in disease. Ven Helmont in comparing the enzymatic tissue reaction to the fermentation action of yeast, was far ahead of his times. However, even during this period the contributions to hepatology were mainly anatomical. Vesalius's (1514-64) *Fabrica* was a sweeping scorn of Galenical and other dogmas and superstitions. The four- and five-lobed livers were set aside among other things. In renaissance physiology the most important chapter is that concerning the circulation of blood. In classical times the liver was the centre of circulation. Michael Servetus (1511-53) stated the hypothesis for pulmonary circulation and his contemporary Realdo Colombo made further advances in circulatory physiology

though he still gave the liver the function of carrying nutritive blood. Cesalpino (1590-1603) gave a general concept of circulation attacking Galen's error of regarding the liver as centre of movements of blood.

17th Century

The 17th century was the great age of specialised anatomic research and notable for a long array of individual discoveries and investigations nearly every one of which had a physiologic significance. The discovery of lacteal vessels in 1622 by Aselli and the thoracic duct and receptaculum chyle by Jean Pequet (1622-74) and the intestinal lymphatics and their connections with the thoracic duct (1651) by Olof Rudbeck (1632-1702), led to the clearing up of the old Galenical error that the veins and the lymphatics of the intestines carried chyle to the liver. The classical description of the distribution and arrangements of the vessels and their fibrous sheaths by Glisson (1654) was the result of a naked eye study of the scraped frame work of macerated specimens of the liver. He missed the significance of the parenchyma. Malpighi (1655) as a comparative anatomist was able to concieve the liver as a conglomorate gland with conical lobules enclosing closely set hexagonal or polygonal labile glandular acini, linked together by their investing membranes and drained by an excretory duct. He also concluded that the portal vein played an arterial role with regard to the liver. He further corrected the earlier misconception of Wepfer regarding the locus of secretion of bile and the direction of its flow and clearly established, by vivisection in kittens, that bile secreted in the acini of the liver was carried down the ducts to the gall bladder and not *vice versa*. Franciscus Sylvius (1614-72) transferred the digestive and blood forming roles of the liver to the newly discovered salivary secretion, attributing to the bile only the supply of the bitter and volatile salts that effervesced with the pancreatic juice in the duodenum.

18th Century

Two of the several chief categories of disease noticed by 18th century medical writers were hepatitis and jaundice which were quite frequent in the daily practice of medicine. The most frequent causes of chronic hepatitis were neoplasms and syphilitic gummata. Eighteenth century concept of hepatitis came into conflict with the symptomatic manifestations of cardiac disease and with the separate category of jaundice. Morgagni (1682-1777) gave the first description of yellow atrophy of the liver ; he also described gummata and cirrhosis of the liver, various calculi and numerous tumours.

Until the 19th century jaundice was believed to be produced by the entrance of fully developed bile into blood, obstruction to outflow of bile being regarded as cause of such entry. Icterus was regarded as the result of reabsorption of bile from intestinal canal. The more obvious symptoms of jaundice such as clayey stools, occasional epigastric pain, pyrexia, etc., were well known. The only form of jaundice considered amenable to treatment was that caused by biliary concretion, patients being bled to relax tension of biliary tract and then forced to vomit to force out the stones.

General symptoms of liver involvement were listed as enlargement, pain during respiration, referred pain in the shoulder girdle, hiccups and vomiting. It was also realised that jaundice was not an invariable sequel ; gall bladder disease was looked upon as a consequence rather as a cause of the liver pathology. The various ways in which the liver abscess might become evacuated were well recognised. The splenic enlargement was generally related to the liver disease. It was long realised that since jaundice might occur in the course of the disease of the spleen as well as of the liver, a relation might exist between the function of the two organs. However, no deep insight into the ultimate causes or relationship between splenic and hepatic enlargements were known except in so far as the concept of hepatitis touched upon the more general one of ascites.

19th Century

Hepatology of the 19th century had two great assets to begin with : Cellular pathology of Virchow (1821-1902) and the use of the microscope in the study of minute anatomy. Kiernan (1833) made classic contributions to the description of portal canals of the liver as containing the portal veins, the hepatic ducts, the hepatic arteries and the vaginal branches of all these vessels with the nerves and absorbents enveloped in a sheath of cellular tissue (Glisson's capsule). His description of circulation in the liver was little modified by subsequent investigations of Theile (1844), Weber (1851) and Beale (1856), and later accounts of Hering (1872), Oppel (1900), Kolliker (1922). The description of the structure of the liver lobule by Kiernan as a 'vascular' or 'hepatic' was opposed by Sabourin (1883) Gerandel and Mall (1906) who contended that the liver was mainly a biliary gland and therefore followed in structure other glands of external secretion. Loeffler and Kretz applied the concept of

Sabourin's lobule in the pathologic field. Kiernan's work on the admixture of arterial and portal blood received support by researches of Conheim and Litten (1876), and later investigations of Pfuhl, Loeffler, Cameron and others. Kupffer (1876) first described cells of the reticulo-endothelial component of the organ while in 1889 Shore and Jones and Renant (1897-99) showed the tubular nature of the glandular elements. Investigations on the lymphatics of the liver were done by MacGillary (1864), Teichman (1887, 1896, 1899), Disse (1890), Wittisch (1874, 1878), Budge (1875), Fleischl (1874) and others. According to the classical concept of lobular structure the polygonal cells of the liver parenchyma were arranged in cord-like fashion. The cord column concept originated with Gerlach (1849) and adopted by Remak (1855), Beale (1856) and Flueger (1869). The limiting plate was recognised by Remak in 1855. The intrahepatic distribution of nervous elements of the liver were studied by Korolkow (1895), Berkeley (1895), Dogil (1895), Wolf (1902). The investigation of the frame work of the sinusoidal capillary net permeating the parenchymal mantle was greatly helped by the application of the silver impregnation techniques of Ramon y Cajal (1852) and Hortega (1851-1918). The multiple vital functions of the liver as a circulatory, haemopoietic, metabolic, digestive, excretory and defensive organ, were slowly revealed. Animal experiments and occasional studies of accidental surgical and pathological occlusion established the hepatic artery as the nutrient vessel of the biliary excretory vessel. The role of the portal vein as the functional vessel of the organ was noted in experiments and observations in atrophic fibrosis of the gland after portal occlusion by Frerichs (1858), Solowief (1875), Doyon and Dufourt (1898), Bainbridge and Leathes (1907). Von Basch (1876) and Mall (1892) also studied portal circulatory functions. Hepatic blood supply and volume output of bile were studied by Ore (1864), Schiff (1870), Betz (1863), Colosanti (1897), Aspe (1900). The present ideas about the liver as an organ of lymph filtration are results of study of minute anatomy by Conheim and Lichthem (1877) on hydraemia and oedema. The role of the liver in sinusoidal endothelium in foetal haemopoiesis was first noted by Kolliker (1846), and later by Weber, Neumann (1874), Schmidt (1892) and others.

In 1843 Claude Bernard discovered that cane-sugar when injected into the veins, appears in the urine but not if treated with gastric juice prior to injection. This was the starting point of his investigations on the glycogenic function of the liver. He published his papers in 1848-50 and by 1857 the glycogenic function of the liver was placed on a permanent basis and he succeeded in isolating glycogen.

Lucien Corvisant (1857-63) had shown that proteids are converted by the pancreatic juice into the ordinary digestive product at the temperature of the body and in alkaline, acidic or neutral media. This corrected Claude Bernard's error that pancreatic proteolysis cannot take place without previous action of bile. Bile derivatives were studied by Thenard (1809), Gmelin (1826). Plattner (1844) obtained crystallised bile and Stecker (1827-71) showed them to be a mixture of sodium salts of glycocholic and taurocholic acids. Austin Flint (1836-1913) claimed that cholesterin is removed from blood by liver and discharged into the body as sterconil (1862), but Naunyn and his pupils assumed it to be a product of liver cells (1892). The common bile tests were introduced by Gmelin (1826), Pettenkoffer (1844), Rosenbach (1876) and Ehrlich (1883). In 1880 Tait introduced hepatotomy.

Original contributions to the study of interstitial hepatitis and cirrhotic jaundice were made by the French clinicians, Georges Hayem (1874) and Victor-Charles Hamot (1875). Naunyn devoted his whole life to the study of liver diseases besides many other diseases. His important contributions were the study of gall stones (1892), the concept of cholangitis, he explained catarrhal jaundice and syphilitic hepatitis, and biliary calculi as the effect rather than the cause of the diseases. He opposed Flint's idea that cholesterin is a specific product of the liver secretion or of metabolism.

Richard Bright left original accounts of acute yellow atrophy of the liver (1836). Thomas Addison's (1793-1860) pills of calomel, digitalis and squills for hepatic dropsy in syphilis are still used. Carl Rokitansky (1804-78) left a classic description of the pathologic appearances in acute yellow atrophy giving the disease its present name (1843). Theodor von Frerich (1819-85) discovered tyrosin in urine in acute yellow atrophy of the liver (1855) and studied the pathology of liver cirrhosis. Murchison (1830-79) who did much valuable work on the liver, himself translated Frerich's book. Laennec (1781-1826) was first to describe 'chronic diffuse interstitial hepatitis'. Ludwig Hektoen has done valuable experimental work in bacillary cirrhosis of the liver (1901). Charcot (1877) left his mark in his lessons on the diseases of the liver and biliary passages. Hepatic pathology made great strides during the period and specialised studies of the appearance of the liver and its associations with other conditions of

the body, were also made. Familial tendencies of juvenile cirrhosis were first emphasised by Jollye (1892). Early references to the association of hepatic cirrhosis and toxic thyroid disease were made by Paul (1865) and Trousseau (1909). Jaundice was first mentioned by Habershon (1874), Eger (1880), Garill (1883), Strumpell (1886), Burton (1888), Westedt (1889), Bonnet and Bourdillon (1891), Noebius (1896), Sutcliff (1898), and Dieulafoy (1901). Mathews Duncan (1879) pointed out that pernicious vomiting in pregnancy is sometimes associated with definite liver lesions, while Pilliet (1888) and Schmorl (1893) considered peripheral lobular necrosis of the liver characteristic of eclampsia, and Abercrombie (1844) described the first case of ruptured liver complicating pregnancy.

Statistical studies of the occurrence of liver diseases in various parts of the world were also begun. Jones (1907) collected three hundred cases of various forms of cirrhosis in children from European and American literature over a period of fifty years. Musser (1899) had another series of 529 cases which included some 400 cases of biliary cirrhosis occurring in India as studied by Ghose.

We hope that this brief sketch of the progress of hepatology through the ages would stimulate more detailed studies of the history of hepatology particularly with reference to India.

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CASE NOTES

ASPIRIN ALLERGY

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Aspirin allergy is very rare but authentic cases of severe collapse and even death after its use have been reported. In allergic individuals quite small doses may cause alarming symptoms.

Case Report

D. H., a 30 year old, Hindu male, was advised on 12-2-56 sulphadiazine 0·5 g. every six hours and aspirin 5 grains in the evening and another five grains next morning for pain in the left ankle joint. As advised, in the evening he took 5 grains of aspirin and immediately after taking the drug he felt itchy sensation all over the body which passed off within an hour. In the morning of 13-2-56 he took another 5 grains of aspirin and shortly after, he noticed development of bullous eruptions and urticaria all over the body. He had also generalised angioneurotic type of oedema specially marked over the right upper limb. He was admitted into the Hospital the same morning.

On enquiry into the past history and family history nothing suggestive could be gathered. No history of taking aspirin-like drugs before could be elicited. The patient was treated with antihistaminics, 50 mg. thrice daily and penicillin injections, 6 lacs units a day for 5 days. Locally lotio calamine with ½ per cent phenol was applied and he was discharged on 21-2-56. Three days before discharge he had swelling of the face with oedema of the lower eyelids but nothing abnormal was detected in the urine and the swelling passed off in the next two days.

Discussion

Aspirin is one of the most valuable pain-relieving drugs very frequently used and so its toxic effects are likely to have in addition an economic significance. However, there are few people who have an intolerance to aspirin serious enough to warrant abstention from it and a very few who are liable to develop really serious allergic reaction and they should carefully avoid the drug which is undoubtedly a difficult task when so many preparations in the market contain the drug.

Allergic reactions are not uncommon after treatment with sulphonamides but they are most likely to occur late in the treatment or on resumption of treatment after an interval, unlike the case under review where the history was very suggestive that it was due to aspirin as he had symptoms immediately after taking the drug and the skin eruptions followed in a few hours.

Summary

A case of allergy due to aspirin is reported. The patient took only five grains of aspirin after which reactions started. He took another five grains after ten hours of the first dose which aggravated the condition.

Acknowledgment

Thanks are due to Drs. M. Khanna and S. Chakravarty of Kulti Hospital for permission to report the case.

SUPPRESSION OF URINE DURING ASPIRIN THERAPY

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Moderate dosage of salicylates may cause diuresis probably due to salt action; large dosage, however, decreases the urinary output (Goodman and Gillman, 1943) probably due to diaphoresis. The following case is one of oliguria during salicylate therapy.

Case Report

C, a 57 years old male, was admitted on 23-7-55 with history of recurrent attacks of joint pains associated with fever of 4 years' duration. On examination the ankle joints were swollen and tender, and the first interphalangeal joint of the left middle finger swollen. The pulse was 72 per minute, temperature 97°F, respiration 20 per minute, B.P.—140/80 mm. Hg, lungs and heart normal.

Laboratory investigations: W.B.C.—6200/c.mm. with polymorphs 60 per cent, lymphocytes 34 per cent, mononuclears 3 per cent and eosinophils 3 per cent; E.S.R.—80 mm. first hour (Westergren); van den Bergh—indirect weakly positive; blood bilirubin—25 mg. per cent, blood urea—36 mg. per cent; blood protein—6 g. per cent with albumin 3·8 g. per cent and globulin 2·2 g. per cent. Urine and stools showed no abnormality.

On 25-7-55: Aspirin 10 gr. t.d.s. was given and within the next 5 days the joint pain almost disappeared.

On 31-7-55: The patient became restless, occasionally with low muttering delirium but responded to questioning. He was thirsty and the skin was dry. The temperature was normal. Urine: albumin—slight, R.B.C. and casts—nil. B.P.—110/65 mm. Hg; pulse—65 per minute. Blood urea—60 mg. per cent. Aspirin was stopped and subcutaneous saline started.

On 1-8-55: No delirium. The tongue was moist and coated; B.P.—100/60 mm. Hg; pulse was 60 per minute and respiration 20 per minute. Blood urea—48 mg. per cent; blood picture—normal; urine—quantity much diminished, otherwise as on 31-7-55.

On 2-8-55: The patient again became delirious. The tongue was moist and clean; pulse rate was 76 per minute and B.P. 108/70 mm. Hg; Urine: passed once in 24 hours, markedly diminished in quantity, otherwise as on 1-8-55 with a specific gravity of 1020. Treatment: Paraldehyde.

3-8-55: Restless preceding night, drowsy during the day. No uriniferous smell of breath. Pulse was 68 per minute. Carbon dioxide combining power—31·9 c.c. per cent; urine about 3 oz. in 24 hours with albumin plus and a specific gravity of 1022.

From 31-7-55 to 3-8-55 the patient received 2 pints of normal saline daily.

From 4-8-55 the patient began to improve. Subcutaneous saline injection pint one a day was maintained upto 5-8-55. Urine passed twice about 10 oz., B.P. 108/66 mm. Hg.; pulse 78 per minute. The tongue was moist. The output of urine increased and there was no albumin in it. The patient maintained steady improvement and was discharged on 23-8-55 with a blood urea—36 mg. per cent, N.P.N.—48 mg. per cent, CO_2 combining power—57·4 c.c. per cent; and B.P. 140/90 mm. Hg.

Discussion

The case under report did not show any other signs and symptoms of salicylate poisoning except oliguria with associated rise of blood urea and fall of blood pressure, the latter occurring markedly during the period of oliguria. The patient had no diaphoresis and dehydration was not the causative factor of oliguria in this case. Low blood pressure was not the precipitating cause either. The urine showed only a small amount of albumin and contained no red cells and casts; renal irritation by aspirin so as to cause impairment of kidney function so much as to produce oliguria was not a likely prime factor. This case had normal B.P. when oliguria developed and urine contained a small amount of albumin without red cells and casts and there was no sweating.

A patient with markedly diminished output of urine, was successfully treated by Miller (1955) on the line advocated by Bull *et al* (1949). He opined that "In a case such as this it is evident that the usual treatment of salicylate poisoning with copious intravenous fluids and alkalies might well prove fatal if continued after the development of severe oliguria". The present case was successfully treated by subcutaneous injection of saline all through.

Acknowledgment

My thanks are due to Dr. H. C. Barua, Superintendent of the Assam Medical College Hospital for permission to report the case and to Mr. B. Bhattacharjee, Librarian of the College, for his help in the preparation of the article.

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INFECTIOUS HEPATITIS

The disease apparently exists all throughout India in an endemic form more commonly in children. It is, however, the recent epidemic of Delhi that has brought it before lime light. This sharp explosive outbreak, which is the feature of water-borne epidemics of infectious hepatitis, has aroused general apprehension.

Such water-borne outbreaks have, however, been reported in the literature on several occasions in the past. For example, an outbreak of jaundice, which followed a few weeks after a water-borne outbreak of bacterial food poisoning, was described in 1931[1]. An explosive outbreak of infectious hepatitis, showing a very high attack rate, was described in 1945[2]. In this epidemic the infection was possibly transmitted by well-water, which showed heavy faecal pollution. In another epidemic, which broke out in some units of the American Army in Italy, it was again well-water[3] which was the probable source of infection. In 1947[4] a water-borne epidemic in guests at a hotel in Sweden was described.

That drinking water can transmit infectious hepatitis has been shown by transmission experiments in human volunteers. Occurrence of infectious hepatitis in these human subjects has furnished convincing evidence of the presence of the causative virus in the water drunk by them. By the same nature of experiments it has also been found that the virus of infectious hepatitis can survive chlorination to the extent of 1 p.p.m. of chlorine for over 30 minutes, and is only somewhat attenuated by 15 p.p.m. The virus also resists heating at 55°C for over 1 hour[5].

It is also these human transmission experiments that leave no doubt that faeces of the infected contain the infective agent[6]. Studies on an outbreak in an orphanage lasting over a period of 8 years also support the view that close contact with faecal material is the common mode of infection[7].

Nevertheless, the mere fact of faecal pollution of water would not necessarily indicate the inevitability of an outbreak of infectious hepatitis. If it were so, Calcutta would not have so far been spared. Seasonal outbreaks of cholera is a regular feature in Calcutta. Both cholera and infectious hepatitis are water-borne. Hence, outbreaks of infectious hepatitis should have been a regular feature too. "The pathogenesis of the disease is by no means clear, and it may be that some activiting factor, such as that met with in infectious hepatitis of mice, is necessary to enable the hepatitis virus to produce its full effect".[8] This activating factor might have been present to cause the Delhi outbreak. Until the exact nature of this activating agent were known, it would be impossible to predict precisely from the detection of faecal contamination alone that an outbreak of infectious hepatitis was imminent.

Excretal contamination of water, especially of large cities, seems to be inevitable. All the water that is excreted, or is used for ablution, bathing, or washing, cannot just be thrown away waste, partitioned off completely and effectively, from the water that is to be used again. The economy of Nature would not permit that; so much fresh water cannot be produced now and then to meet the needs of man. What, however, could be done with human ingenuity is to make the excreta as inocuous as possible, and dilute it so well that water contaminated with these still remains potable. This needs a team work of public health bacteriologists, chemists, engineers, and administrators, and the public at large. The levels of safety of drinking water, and the methods of ensuring such safety, can be laid down by the bacteriologists, but the enforcement of these criteria is a more general responsibility. Prevention of dangerous contamination is as much a public responsibility as that of public health personnel. If all the cholera stools, or the faeces of those suffering from infectious hepatitis, were thrown away without prior disinfection into the gutter, the community has naturally to blame itself for its doom.

Prevention of water-borne epidemics should be easy if dangerous faecal pollutions could be avoided by all means. It is futile to depend on senses

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of taste and smell to detect such contamination. Bacteriological and chemical tests are available for detection of faecal contamination of water. However, it should be remembered that such tests are not infallible. Moreover, mere absence of evidence of faecal pollution does not necessarily indicate that pollution has not taken place. All it does show is that at the time the sample was examined, there was no detectable evidence of pollution. It is the enforcement of scrupulous sanitation, encouragement of general hygiene, and frequent checks on sanitary engineering devices, that are better safeguards against water-borne epidemics, than mere bacteriological and chemical tests on water-pollution.

Spread of outbreak of infectious hepatitis can also be prevented by mass-immunisation with gamma-globulin. The prophylactic effect of gamma-globulin on infectious hepatitis has been reported in the past, as well as very recently[9, 10]. As little as 0·01 ml. per lb. body weight given intramuscularly during the incubation period may apparently prove effective. There is, however, a dangerous drawback of such immunisation. The virus of serum jaundice may be present in the blood used for the preparation of the gamma-globulin. Use of this virus-containing gamma-globulin would cause great scare by virtually producing the jaundice which it was claimed to prevent. It, however, appears that the gamma-globulin prepared by the ethanol process is free from the virus[11]. Ethanol precipitated gamma-globulin would, therefore, appear to be a safe prophylactic. It is, however, of no value for the treatment of established disease[12].

This prophylactic value of gamma-globulin would naturally suggest the need for preparation of gamma-globulin for prevention of future outbreaks, and it has entreated the Blood Banks of India to take up this task[13]. The magnitude of this task should, however, be appreciated before proceeding with such an ideal.

A skin test has recently been devised[14] for differentiating the immunes from the non-immunes. For this test, an antigen prepared by irradiation of infected amniotic fluid is used. A positive reaction is produced in about 50 per cent of those suffering from the disease, while between 90 and 100 per cent of those recovered show positive reaction. The test, thus, reveals presence of hypersensitivity which is very closely associated with immunity.

No figures for proportionate distribution of the immunes and non-immunes in a given population are yet available in this country. Yet, the large incidence in the Delhi outbreak would suggest that quite a considerable number of non-immunes would prevail in any city. Hence, in a city like Calcutta, perhaps about one million persons would need immunisation, assuming that three-fourths of the population are immunes while only the remaining one-fourth would need immunisation. The average dose of gamma-globulin, which is required to immunise the non-immunes, would not be less than 1 ml. per person. Hence, about one-million ml. of gamma-globulin would be required for launching a mass-immunisation campaign in Calcutta.

Ethanol precipitated Fraction II of Cohn[15], which consists of gamma-pseudoglobulin—the richest carrier of immune bodies—represents approximately 5 per cent of the plasma proteins. And plasma constitutes only about half the volume of the blood. Hence, 40 ml. of blood would yield only 1 ml. of gamma-globulin of adequate concentration. On the basis of this approximate calculation, over 40 million ml. of human blood would be required to meet one immunisation campaign in Calcutta.

It should also be taken into account that immunisation with gamma-globulin is as short-lived as passive immunisations would be expected to be. However, the human gamma-globulin, being homologous protein, would last for about 6 months. Hence, if an epidemic threatens again after the lapse of this period, another 40 million ml. of human blood would be required!

Would this appear to be a possible proposition in a welfare country where mass-immunisation with such inexpensive materials as tetanus and diphtheria toxoids has not yet been possible? Yet, it is a well-known fact that the toxoids induce immunity of the longest duration much longer than that obtained with all passive immunising agents and most of the active immunising agents too.

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CURRENT MEDICAL LITERATURE

Effective Oral Antidiabetic Drug (BZ 55)

BERTRAM AND OTHERS (*German M. J.*, **1**: 8, 1956) give in the following lines the summary of their observations on the clinical trials of an effective oral antidiabetic drug (BZ 55):

A new hypoglycaemic drug, sulfanilyl-n-butyl-urea (BZ 55) was used in 82 patients with diabetes mellitus of differing severity and duration of illness. All had been carefully observed for some time. Frequent blood sugar determinations, glucose tolerance tests and urine examinations were performed before and during the trial period. BZ 55 was administered in tablets containing 0·5 g. of the drug. Five tablets were given on the first day, 3 on the second, and 2 daily thereafter. At this (and even higher) dosage, young diabetics showed practically no response. But elderly ones had remarkable results. One group, consisting of 28 patients between the ages of 49 and 77 years who had been inadequately treated or not at all, showed the best therapeutic response. All but 3 showed a rapid decrease of blood sugar levels to below 150 mg. per cent. In several patients the drug could even be discontinued after 10 days without relapse during a further observation period of 3 months, as long as strict diet was maintained. If relapse occurred, renewal of treatment was again successful. Response in 3 patients with mild acidosis was poor, despite high doses. Evidently, the drug is not suitable for sudden metabolic disturbances. Another group of 38 patients, between the ages of 41 and 77, had previously been on insulin, some for many years. Complete replacement of insulin was achieved in 28. No harmful or unpleasant side-effects were ever observed. The mode of action is thought to be on the α (alpha)-cells of the pancreas by decreasing their production of an hyperglycaemic, insulin-antagonistic substance. It is stressed that further careful studies are still needed, before routine use of the drug can be recommended in the elderly diabetic.

Brilliant Green Aerosol in Pulmonary Moniliasis

SMITH AND ARMEN (*Ann. Int. Med.*, **43**: 1302, 1955) give a brief review of the literature of pulmonary moniliasis.

The authors maintain that the most reasonable and reliable set of criteria for diagnosis is the *persistent cultural isolation* of the fungus from bronchial washings as well as from sputum *in the absence* of other possible casual agents in a patient with both clinical and radiological evidence of pulmonary disease.

They present a case of severe pulmonary moniliasis which responded very well to brilliant green aerosol.

The treatment was begun with 2 c.c. of 0·1 per cent brilliant green in 50 per cent propylene glycol by oxygen aerosol inhalations five times daily. This was raised to 0·2 per cent after 6 days and was well tolerated. If this were completely absorbed it would represent a daily dose of 0·25 mg/kg within the safe range. The patient received many interrupted two to four-week courses of treatment. After one year the patient reports that he continues to gain weight, works eight hours a day during the week and about his house on weekend, has a mild productive cough but feels very well.

Metacortandracin in Disseminated Lupus Erythematosus

STEINBERG AND ROODEMBURG (*Ann. Int. Med.*, **44**: 316, 1956) give in the following lines the summary of their observations:

Nine patients, six with disseminated lupus erythematosus and three with periateritis nodosa, have been treated with metacortandracin. One patient with periarteritis nodosa died while under treatment. The autopsy showed extensive arterial involvement, both visceral and peripheral. The most remarkable feature in the histologic study was the lack of inflammatory process noted in the diseased arteries. The inference from these studies is that, if this patient had been treated in the early phase of the disease, the outcome would have been more favourable. The other two cases of periarteritis nodosa have been converted from very ill people to a status of employability.

All six patients with disseminated lupus erythematosus had previously been treated with either cortisone or corticotropin. All have done much better with metacortandracin. However, in no instance have the L.E. cells disappeared either from the bone marrow or from the peripheral blood. The improvement in the blood picture in case 1 was marked. All six cases carry on their usual activities with little or no restriction. The capacity to tolerate a normal diet, without salt restriction or the addition of the large, gastric-disturbing doses of potassium required with the use of other steroids is appreciated by these people.

The initial dose was 30 mg.—10 mg. every eight hours in all cases except case 7. The dose was decreased by 5 mg. every five days until the smallest amount required for maintenance was reached. It was usually 15 to 20 mg. per day.

All of these patients with the exception of case 5 (L.E.) have been observed under treatment for from 60 to 120 days. This is still a short time although the short-term treatment has been favorable, more time will have to elapse before a conclusive opinion can be reached as to long-term treatment of the collagen diseases with this new compound.

A Guide to Antibiotic Therapy

VALENTINE (*Practitioner*, **176**: 62, 1956) gives in a tabular form a guide to antibiotic therapy.

Abbreviations: pen.—penicillin; str.—streptomycin; tetra.—one of the tetracyclines (chlor-, oxy- or tetracycline); chl.—chloramphenicol; erythro.—erythromycin; sulph.—sulphonamide; sens.—sensitive; rt.—resistant; I/M—intramuscular; I/V—intravenous; I/T—intrathecal.

Condition and Type	Drug, Dosage and Course	Remarks
Staph. infection		
(1) Generalized	(1) Pen. 0·5 mega t.d.s. for 3 weeks if sens. If rt. to pen. tetra. 0·5 g. 6-hourly, or erythro. 0·5 g. 6-hourly, or both.	(1) Sens. test essential. Surgical treatment of abscess, etc., if present.
(2) Cutaneous	(2) Test sensitivity: in local treatment avoid drug capable of systemic use.	(2) Disinfect skin round lesions with spirit or cetrimide until healed. Need for systemic treatment varies with case and lesion.
(3) Superinfection, e.g. enteritis	(3) Erythro. 0·5 g. 6-hourly for 5 days, or parenteral 0·25 g. 6-hourly.	(3) Fluid replacement. Stop existing antibiotic therapy.
Upper respiratory tract infection		
(1) Sore throat	(1) Procaine pen. 0·3 mega for 7-10 days, or sulph.	(1) Drugs only needed in severe cases of infection with *Str. pyogenes* or old rheumatic fever case.
(2) Otitis media	(2) Pen. 0·5 mega at once, then oral pen. if necessary.	
(3) Acute sinusitis	(3) Pen. 10,000 units/ml. in spray or drops with fever, also pen. I/M.	
Pneumonia		
(1) Pneumococcal, *Str. pyogenes*	(1) Pen. aqueous + procaine, 0·3 mega/day, or sulph. full dose.	Culture if possible to detect unusual types, especially during influenza epidemic and with poor response. Duration of treatment varies with case.
(2) *H. influenzae* post-influenzal	(2) Tetra. 1 g. daily.	
(3) Staphylococcal	(3) If sens., pen. 0·5 mega t.d.s., or tetra. or erythro. 2 g./daily.	
(4) Friedlander	(4) Str. 2-3 g./day, or tetra. 1-2 g./day.	
Bronchial infection		
(1) (a) Chronic	(1) (a) Prolonged treatment inadvisable.	(1) Infection usually pneumococcal or *H. influenzae*.
(b) Acute exacerbation ...	(b) Pen. or tetra, as in pneumonia.	
(2) Pertussis	(2) Tetra. 35 mg./kg.	(2) Of value during first week only.
Endocarditis		
(1) *Str. viridans*	(1) Pen. 2-4 mega/day for 4-6 weeks.	(1) If pen. and str fail, tetra-pen. rarely successful.
(2) *Str. faecalis*	(2) Pen. 10-20 mega/day—str. 2 g./day for 6 weeks.	(2) Tetra., chl. and erythro. useless.
Acute meningitis		
(1) Meningococcal	(1) Sulphadiazine or sulphatriad full dose.	All forms: continue 1 week after C.S.F. sterile. (1) If very severe, add pen., 1 mega 2-hourly I/V.
(2) Pneumococcal (or *Str. pyogenes*)	(2) Pen. 1 mega I/M 2-hourly or 0·5 mega I/M t.d.s.—20,000 units I/T b.d.s. (addition of tetra. or chl. contraindicated).	(2) With patient sensitive to pen., chl. 100 mg./kg. daily may succeed.
(3) *H. influenzae*	(3) Chl. 100 mg./kg./day, or str. 45 mg./kg. I/M—50-100 mg. I/T daily sulph.	(3) C.S.F. level of 10 ug./ml. of chl. needed. With vomiting, str. better.

Condition and Type	Drug, Dosage and Course	Remarks
(4) *Others*	(4) Test sensitivity.	(4) Bacitracin, neomycin and polymyxin may be given I/T.
Alimentary infections		
(1) Enteric fever	(1) Chl. 2 g./day for 4-5 days, then 1 g./day for 7-10 days.	(1) Chl. useless in faecal carrier.
(2) Bacillary dysentery ...	(2) If sens. sulphadiazine or phthalylsulphathiazole. If rt. to sulph., oral str. 3 g./day or tetra. 1-1·5 g./day.	
(3) Infantile gastro-enteritis ...	(3) Isolate pathogen and test.	(3) Fluid replacement essential.
(4) Peritonitis	(4) Tetra. 1-2 g./day I/V, or pen. 2 mega-str. 2 g./day I/M.	(4) Also fluid and electrolyte replacement.
Urinary infections		
(1) *E. coli*	(1) Alkali and sulph., during culture and sensy. test.	Infection, especially if recurrent, often due to obstruction or other abnormality. Effect of drugs only temporary and follow-up essential.
(2) Gram + cocci	(2) Pen. usually.	
(3) Proteus	(3) ? Pen. 0·5 mega t.d.s. ? 'Furadantin 5-8 mg./kg.	(2), (3) and (4) Antibiotic therapy should be limited to severe, intractable infections, and to cover surgery.
(4) *Ps. aeruginosa*	(4) ? Str. 2-3 g./day for 2 days. ? Polymyxin B or E 1·5-2 mg./kg./day, in 4-hourly I/M dose.	
Actinomycosis	Pen. 0·5 mega. t.d.s. for 4 weeks or more, or tetra. 1-1·5 g./day, ? 4 weeks.	Str. may be combined with pen. Chl. also reported effective.
Anthrax	Pen. 0·5 mega b.d. for 5-7 days, or tetra. 1-1·5 g./day for 5-7 days.	Chl. also effective.
Gas gangrene	Pen. 1 mega t.d.s.	Surgical toilet of wound remains, essential. Also give antitoxin.
Erysipeloid	Pen. 0·5 mega b.d.s.	
Weil's disease	Pen. 0·5 mega t.d.s., or tetra. 1-1·5 g./day.	Treatment often ineffective in severe cases.
Brucellosis	Tetra. 1-2 g./day for 2-4 weeks.	Addition of str. 1-2 g./day said to reduce relapse rate.
Tularaemia	Str. 1-2 g./day for 7 days, or tetra, 1-2 g./day for 7 days.	
Plague	Str. 2-3 g./day ? 5-7 days, or ? tetra. 2 g./day for 7 days.	Sulph. less effective but of value in prophylaxis. In severe cases also give antiserum if available.
Rickettsial infection		
(1) Scrub typhus	(1) Tetra. or chl. 6 g. in 24 hours, repeat if necessary.	Drugs only suppress disease; cure due to immunity after 2nd week.
(2) Other forms	(2) Tetra. 50 mg./kg./day, or chl. 50-100 mg./kg./day.	(2) In severe cases tetra. I/V 5-10 mg./kg./day at first; course of 4 or more days. Effect least striking in Q fever.

Condition and Type	Drug, Dosage and Course	Remarks
Virus		
(1) Psittacosis—L.G.V. group ...	(1) Tetra. 1-1·5 g./day, course varies, or chl. 2-3 g./day. (2) Ineffective, except in control of secondary infection.	
Venereal disease		
(1) Syphilis	(1) (a) Procaine pen. 0·6 mega/day for 10 days.	All forms; treatment by expert advised.
(a) Early		(1) In special circumstances benzathine pen. 1·2 mega, single dose.
(b) Late	(b) As above for 10-20 days (? bismuth in addition.)	
(2) Gonorrhoea	(2) Procaine pen. 0·3 mega, one dose.	(2) Str., tetra., chl. and erythro. also effective. Many strains now resistant to sulph. In chronic case response often poor.
(3) Lmphogranuloma venereum	(3) Tetra. 1·5-2 g./day for 5 or more days, or sulph. 5 g./day for 10-14 days.	(3) Chl. also effective. Buboes may need aspiration. Pen. and str. useless.
(4) Granuloma inguinale ...	(4) Str. 3-4 g./day for 5 days, or tetra. 1·5-2 g./day.	
(5) Chancroid	(5) Sulph. 5 g./day for 10-14 days.	(5) Str., tetra. and chl. also effective.
Other spirochaetal infections e.g. yaws, pinta, bejel	Procaine pen.—2 per cent aluminium stearate 1·2 mega; repeat at 4-5 days, or benzathine pen. 1·2 mega, single dose.	

Rheumatic Fever Pneumonitis

LUSTOCK AND KUZMA (*Ann. Int. Med.*, **44** : 337, 1956) from a clinical and pathologic study of 35 cases observe :

Autopsy material from patients dying of rheumatic fever at the Milwaukee Children's Hospital, the Milwaukee County Hospital and the Veterans Administration Hospital and some private patients was studied. Thirty-five instances of rheumatic fever pneumonitis were discovered, and the pathologic material was reviewed. The clinical aspects of these cases were studied in retrospect.

The diagnosis of rheumatic fever pneumonitis is rarely made ante mortem. The seriousness of this complication of rheumatic fever warrants clinical interest directed toward more successful management. This is predicated on more frequent recognition of rheumatic fever pneumonitis during the acute phase of the disease.

Clinical diagnosis of rheumatic fever pneumonitis is warranted when there is :

A. Disproportionate respiratory distress with severe cough, chest pain, cyanosis and haemoptysis not relieved by oxygen and the customary supportive measures.

B. Evidence of carditis, but not of sufficient severity to explain the pulmonary findings, in the presence of prolonged high fever and negative blood cultures which do not respond to salicylates.

C. Chest x-ray finding of increased perivascular markings arising at the hilus and progressing to nodulation, confluence and massive consolidation with relatively clear apices and bases.

Gross pathologic changes are : rubbery consistency, various dark hues of focal haemorrhages, fine granularity and spotty vesicular emphysema.

Histologic changes are : alveolar haemorrhages, necrotising alveolitis, hyaline membranes, alveolar lining cell proliferation, organisation of exudate, fibrinoid necrosis of bronchiolar lamina propria and arteritis.

Electroencephalographic Pattern in Hepatic Coma

BICKFORD AND BUTT (*J. Clin. Invest.*, **34** : 790, 1955, Ref. *Abs. World Med.*, **13** : 67, 1956) from the analysis of the electroencephalographic changes in 13 patients in hepatic coma in the Mayo Clinic observe :

The earliest change was a slowing and disorganisation of the alpha rhythm. The appearance of theta activity was accompanied clinically by clouding of consciousness and later, when the patients could be roused only by painful stimuli, triphasic waves appeared. Complete recovery within a few hours occurred in some cases, but otherwise generalised delta activity appeared, which finally showed a gradual suppression.

Triphasic waves were demonstrated in 12 of the 13 patients and were usually best seen in the frontal and temporal areas. They were symmetrical, the main deflection was positive, and they were unaffected by

afferent stimulation. In the occipital regions there was a delay of 25 to 140 milliseconds, suggesting that the triphasic waves were conducted—but whether by cortical or subcortical mechanisms was not clear. Similar discharges may sometimes be seen in patients under ether anaesthesia, in epileptics, and in cases of cerebral lipoidosis. The EEG recordings from 8 patients suffering from cirrhosis of the liver but without mental changes were all normal.

Amiphenazole and Morphine in Intractable Pain

McKeigh and Shaw (*Brit. M. J.*, **1** : 142, 1956) in reporting on the treatment of 127 cases of intractable pain in terminal carcinoma observe :

Complete abolition of pain was obtained for 24 hours by the use of large doses of morphine (1-3 gr., 65-195 mg.) and a new drug, amiphenazole. The mixture was given three or four times a day for periods of up to eight months. The main advantages of the combination are as follows : Administration of large amounts of morphine without respiratory depression, nacrosis, or depression of the cough reflex; amiphenazole apparently prevents the onset of any marked tolerance to morphine, and possesses a central nervous stimulant action of the caffeine type; and treated patients have bright mental outlook under otherwise hopeless conditions.

Three Types of Human Diabetes

Lawrence (*Ann. Int. Med.*, **43** : 1199, 1955) gives in the following lines the summary of his observations on three types of human diabetes :

Three types of human diabetes, all showing hyperglycaemia and glycosuria, are described and differentiated (1) on simple clinical grounds of excess or lack of stored body fat, and by the presence or absence of ketosis, and (2) by experimental work showing the different amounts of active insulin in their blood or extractable from the pancreas after death.

Lipoplethoric diabetes, the commonest form, is found in the obese middle aged, *without ketosis* and with a normal, or nearly normal, blood and pancreatic insulin content. Reducing diet controls the condition. Any intercurrent infection produces a heavy ketosis requiring the temporary use of insulin to which it quickly responds. Later on especially in the poorly controlled, the common complications develop—retinopathy, nephropathy and vascular lesions.

Insulin deficient diabetes is also a common form, especially in the young, with loss of weight, *heavy ketosis*, no plasma insulin and little pancreatic insulin. Insulin treatment is essential.

Lipo-atrophic diabetes. Five cases of this very rare syndrome in young people are described. It is differentiated clinically by a complete absence of body fat, by an intense hyperlipaemia, hyperglyaemia, no ketosis but insulin-resistant; hepatomegaly with ultimate portal cirrhosis; a raised basal metabolic rate.

The fundamental differences of these three types of diabetes mellitus are discussed.

B.C.G. and Vole Bacillus Vaccines

The following is the summary of the first (progress) report of the M.R.C. by their tuberculosis vaccines clinical trial committee :

A controlled clinical trial of B.C.G. and vole bacillus vaccines in the prevention of tuberculosis in adolescent boys and girls started in September, 1950. By December, 1952, approximately 56,700 volunteers, all in their final year at secondary modern schools in or near North London, Birmingham, and Manchester, had been included; nearly all were aged between 14½ and 15 years. Those found at an initial radiographic examination to be suffering from tuberculosis, and those known to have been in recent contact with a case of pulmonary tuberculosis at home, were excluded from the trial. This first report presents preliminary results after each participant had been in the trial for two and a half years, with supplementary incomplete information up to four years.

At the initial examination, each entrant had a chest radiograph and an intracutaneous test with 3 T.U. (tuberculin units); those with negative reactions to 3 T.U. were tested with 100 T.U. Those negative to both strengths were allocated by a random process to an unvaccinated, a B.C.G.-vaccinated, or a vole-bacillus-vaccinated group. The participants were thus automatically classified on entry into the following five groups : tuberculin negative, left unvaccinated (13,300 entrants); tuberculin negative, B.C.G. vaccinated (14,100); tuberculin negative, vole bacillus vaccinated (6,700); tuberculin positive to 3 T.U. (16,000); and tuberculin positive to 100 T.U. but not to 3 T.U. (6,600). Vole bacillus vaccine was not used in the London area, and was not available for all of the time in the Birmingham and Manchester areas. Many of the volunteers had a second examination (similar to the first) three to five months after entry, while they were still at school. No participant was vaccinated or revaccinated by the investigating teams subsequent to the examination on entry.

After leaving school, participants in each of the five groups have been followed with similar intensity by means of a fourteen-month cycle of inquiry and examination, each cycle consisting of a postal inquiry, a home visit by a health visitor, and an examination which, as before, included a chest radiograph and tuberculin tests. As a result, contact was made with 94 per cent of the participants by at least one of these three means within eighteen months of entry; information has since been obtained from many of the remaining 6 per cent. In addition to these methods of discovering the cases of tuberculosis which arose, information from notification lists of medical officers of health and from chest clinic records was also made available.

All definite and suspected cases of tuberculosis have been reviewed and classified by an independent assessor, who, to avoid bias, was kept unaware both of the results of all the tuberculin tests and of whether any vaccination had been performed. A total of 165 definite cases began within two and a half years of entry to the trial. Of these, 63 per cent were of pulmonary tuberculosis and 22 per cent of pleural effusion without evidence of pulmonary tuberculosis; 68 per cent of the cases were

severe enough for the patients to be taken off work for at least three months. There was no death from the disease during the two and a half years.

The annual incidence of tuberculosis in the tuberculin-negative unvaccinated group was 1·94 per 1,000; in the B.C.G.-vaccinated group it was only 0·37 per 1,000; and in the vole-bacillus-vaccinated group only 0·44 per 1,000. (Strictly this last figure should be compared with the incidence among those participants in the Birmingham and Manchester areas who were admitted concurrently with those given vole bacillus vaccine—namely, with 2·06 per 1,000 in the negative unvaccinated group and 0·31 per 1,000 in the B.C.G.-vaccinated group). Each vaccine therefore conferred a substantial and similar degree of protection against tuberculosis over a period of two and a half years in adolescence. The strength of the earlier batches of vole bacillus vaccine was below the standard intended.

The protection conferred by each vaccine was evident soon after it had been given, and was still substantial between two and two and a half years after entry. Supplementary incomplete information up to four years suggests that the protection is maintained for this period. Although the numbers of cases in the vaccinated groups were small, the evidence does not indicate that protection was limited to tuberculosis in particular sites, nor that the pulmonary lesions were less extensive or severe in those who had been vaccinated but developed the disease.

Complications of vaccination consisted of occasional regional adenitis and delayed healing of the local lesion. Two cases of erythema nodosum were also attributed to B.C.G. vaccine. In addition a number of those given vole bacillus vaccine developed lesions, indistinguishable from lupus vulgaris, at the site of vaccination; up to the end of June, 1955, 22 of these had required treatment.

Among the entrants with a positive reaction to 3 T.U. the annual incidence of tuberculosis was 1·75 per 1,000, compared with 0·74 per 1,000 among those positive only to 100 T.U. The annual incidence was particularly high among those with strong reactions to 3 T.U. on entry (15 mm. induration or more)—namely, 2·93 per 1,000, compared with 0·78 per 1,000 among those with 5-14 mm. induration. Thus, in this age group those highly sensitive to tuberculin appear to have a special risk of developing tuberculosis.

The annual incidence of 0·74 per 1,000 among those positive only to 100 T.U. compares with 1·94 per 1,000 in the concurrent negative unvaccinated group. These results are not those which would be expected if positive reactions to 100 T.U. only were non-specific for tuberculous infection. The interpretation of weak reactions to tuberculin requires further investigation.

If no participant in the present trial had been vaccinated, a total of 246 cases of tuberculosis would have been expected within two and a half years of entry; if all the tuberculin-negative entrants had received B.C.G. vaccine a total of 111 would have been expected. This represents an expected reduction of 55 per cent in the total incidence of tuberculosis for the two and a half years. However, 134 cases of previously unsuspected definite tuberculosis which were present on entry were excluded from the trial, nearly all as a result of the initial radiographic examination. In the absence of this radiograph, many of these cases would apparently have arisen after entry, and the apparent reduction in the total incidence of tuberculosis would have been only of the order of 35 per cent.

The implications of these interim findings for the use of vaccination in the control of tuberculosis in adolescents are discussed. The trial is still in progress, and later reports will contain more detailed analyses over longer periods of time.—*Brit. M. J.*, **1**: 413, 1956.

Factors Influencing Relapse in Pulmonary Tuberculosis

OYAMA (*Am. Rev. Tuberc.*, **72**: 613, 1955) writes:

Of 2,571 patients discharged from a certain sanatorium from 1948 through 1953, 1,534 patients were found to have an uncomplicated reinfection type of pulmonary tuberculosis that could be adequately evaluated. Adequate follow-up reports were obtained in 1,259 patients observed for periods ranging from 11 months to six years. The cumulative incidence of reactivation during observation following discharge was 28·5 per cent.

It was found that the factors resulting in relapse in pulmonary tuberculosis are so complicated that one factor may be analyzed alone without finding any apparent association with the rate of reactivation, but, if combined with another factor, it may be found to have a significant correlation with the incidence of relapse.

This study revealed that the post-treatment relapse rate for pulmonary tuberculosis was definitely higher in males than in females. However, there were no important differences in relapse rates for the two sexes in certain age groups, e.g., 10 to 29 and over 70. Patients in the older age groups showed a greater tendency to relapse. This may possibly be explained by the fact that the older age groups had less adequate total treatment during the acute phase of tuberculosis. Furthermore, their age would often make resectional surgery unfeasible.

The type of treatment appeared to influence significantly the incidence of reactivation. It was evident throughout this study that prolonged antimicrobial therapy for one year or more was remarkably effective, as was pulmonary resection combined with antimicrobial therapy. Resectional surgery plus antimicrobial therapy was superior to antimicrobial therapy alone in patients with far advanced tuberculosis, but this was not true in patients with moderately advanced tuberculosis.

There were no significant correlations between the incidence of relapse and: (1) right or left pulmonary lesions among unilateral cases, (2) duration of disease, (3) length of hospitalization, (4) duration of "bed rest" alone, and (5) the particular regimens of antimicrobial therapy when used alone.

PAS and INH Concentrations in Sputum

SATO AND TAYAMA (*Rep. Research Inst. Tuberc. and Leprosy*, Tohoku, **6**: 309, 1955) give below the summary of their observations:

PAS and INH concentrations in sputum from patients receiving the drug were chemically determined.

(A) PAS.

(1) Single oral dosage of 10 g. : Course of PAS concentration in sputum made one peak with values of 6·6 to 27·9 γ per ml. for free and 7·28 to 33·6 γ per ml. for total PAS 4 hours after administration. These values were one-thirteenth of that in blood for both free and total PAS. Free PAS was not detected after 6 hours, but conjugated PAS was detected till 12 to 24 hours after the administration though it was very small in amount.

(2) Oral administration of 10·5 g. divided in three times :

The course of PAS concentration in sputum was zig-zag with peaks after each dosage; the highest peaks with values of 3·3 to 7·4 γ per ml. for free and 5·9 to 9·8 γ per ml. for total PAS were seen about 3 hours after the second intake. These values were one-seventeenth for free and one-thirteenth for total PAS that in blood. In the following morning free PAS was scarcely found in sputum but conjugated PAS was found in amount of 2·1 to 10·6 γ per ml. The time of sputum peak generally coincided with that of blood.

(B) INH.

(1)Single oral dosage of 3·3 to 3·75 mg. per kg. :

The course of INH concentration in sputum made a parallel curve with that in blood, its sputum peaks of 2·7 to 3·1 γ per ml. seen after 3 hours were slightly lower than that in blood. After 7 to 13 hours INH was not detected in sputum.

(2) Dosage given in three divided doses :

The maximum sputum levels of INH were 2 to 2·8 γ per ml. when total doses of 3·75 to 6·7 mg. per kg. were given in three divided doses. The time at which the maximum levels were attained varied from case to case, but as a whole courses of sputum level were like those of blood.

In conclusion, it may be said that PAS and INH are excreted in sputum in quantity sufficient enough to be bacteriostatic for tubercle bacilli though its level is very low compared to that in blood in the case of PAS. The question that these chemotherapeutics present in sputum might act as bacteriostatically on cultivation of tubercle bacilli is unlikely from the figures obtained above, for the drugs are highly diluted during per-treatment, such as lavage, of sputum.

Disturbance in Acid-Base Balance after Pulmonary Resection

NAKADA (*Rep. Research Inst. Tuberc. and Leprosy*, Tohoku, **6** : 273, 1955) gives below the summary of his observations on the disturbances in acid-base balance after pulmonary tuberculosis :

In pulmonary resection under general anaesthesia, the disturbances in acid-base balance in blood are such that a slight respiratory acidosis is caused immediately following operation, but the awakening from general anaesthesia, the rise of body temperature and the quickening of respiration bring about a tendency towards respiratory alkalosis, which gives place to respiratory acidosis again approximately 24 hours after operation, complicated with metabolic acidosis. Up to this period, the disturbances in acid-base balance in blood generally correspond with clinical symptoms, but 3-5 days after operation when metabolic acidosis is complicated with very severe respiratory acidosis clinical symptoms are not always remarkable. This is the period that requires the greatest care in the postoperative treatment of pulmonary resection. Ten days after operation, all the factors in acid-base balance are restored to their normal values and are stabilized.

It is considered that about the same alterations are brought about in cases of pulmonary resection under local anaesthesia, though the respiratory acidosis is not remarkable immediately or several hours after operation.

Continuous pernasal oxygen inhalation is a very effective treatment for postoperative anoxic anoxia, though it is accompanied with a slight anoxia 18 hours, 2 days or 7 days after operation. Appropriate blood transfusion is of vital necessity for the severe anaemic anoxia which is evidenced 7 days after operation.

CURRENT TOPIC

POSTGRADUATE MEDICAL EDUCATION IN ISRAEL

Six medical institutions are taking part so far in postgraduate medical education in Israel :

(1) In the first place, the Medical Association of Israel, which since its foundation over 40 years ago, has considered postgraduate education of the physicians, immigrated to Israel from all parts of the world as its chief task and has created a special instrument for the organisation of this work in the form of the "Advisory Council". In the main towns, a Scientific Secretary, jointly with a Scientific Advisory Board, organises the scientific weekly meetings of the Society, clinical visits or case presentations in the hospitals, and evening or week-end courses in electrocardiography, liver diseases, haematology, etc. The Advisory Council arranges regular meetings, lectures and symposia in the smaller branches throughout the year. In the lectures, all the institutions and hospital centres of the country are represented. They include almost every field of preclinical and clinical medicine, such as : diagnosis and treatment in emergency cases; drug treatment in cardiac cases; allergic diseases; some problems of the diabetes clinics; evaluation of the usefulness of some modern laboratory methods for the general practitioner; hormonal treatment in gynaecology; psychosomatic aspects of internal diseases, etc.

The Advisory Council elects the President of the biennial Scientific Conventions of the Israel Medical Association and offers suggestions on the main topics. Jointly with other scientific bodies, it organises at certain intervals, symposia on border topics, where scientists and clinicians discuss the problems for their different points of view (for instance : adrenal cortical hormones; chemistry, physiology, clinical aspects; thyroid hormones, etc.).

The Advisory Council recommends the candidates for postgraduate studies abroad. The branches of the Israel Medical Association range from Nahariа and Tiberias in the North to Beersheba in the South, including the Arabic township of Nazareth. (The lectures are there given in English, in all the other branches in Hebrew).

(2) The Central Workers' Sick Fund (Kupat Holim) with its 1,300 physicians, large modern hospitals and outpatient-clinics provides the medical care for almost 2/3 of the whole population, in particular almost the entire rural population. The postgraduate education of its physicians is a task of particular importance, since these physicians constitute the bulk of all the general practitioners in the country and almost all the physicians in villages and settlements. The physicians of Kupat Holim have created special institutions for postgraduate education. Its Postgraduate School for Medical Studies is affiliated with the Beilinson Hospital in Petah Tikva: For 5 years already postgraduate courses of 6 weeks' duration are given to groups of 12-20 general practitioners, including lectures, pathological, clinical and x-ray demonstrations, bed-side teaching, study of medical literature in the library, etc. Similar to the National Health Service in England, 4 weeks of the course are financed by the Sick Fund, the participating doctors receiving board and lodging at the hospital.

(3) The Ministry of Health is actively interested in the postgraduate education of Israel's physicians and conducts joint staff conferences, visits at their hospitals (open to every physician in the country) as well as special courses in Public Health.

(4) The Malben Organization, which set itself the task of caring for chronically ill among the new immigrants, has made special arrangements for postgraduate education in tuberculosis, geriatrics, chronic nervous diseases, etc.

(5) The Municipality Hospital Tel Aviv—Jaffa offers special courses in postgraduate education.

(6) Last but not least, the Hebrew University Hedassah Medical School, opened in 1949, has exercised a marked influence on medical postgraduate education through regular clinical conferences in the University Hospital and other departments. Since November 1, 1955, there exists in the framework of the Medical School a special department for postgraduate studies, under the guidance of an Executive Secretary. It is the task of this department to study the needs and possibilities of this country in postgraduate education and to work out a plan for the setting up of a School of Postgraduate Studies in the framework of the Medical School, in close co-operation with the above-mentioned institutions which have already gained experience in this special field of education. The aim of the project is to mobilize the best teachers in the country and to raise postgraduate medical education to a high academic standard. The courses recently organized by the new department are a beginning in this direction, such as a course on diabetes of 21/2 days' duration at the postgraduate institute of Beilinson Hospital, conducted by 6 teachers of the Hebrew University, including the Dean of the Medical Faculty, 5 teachers of the Kupat Holim Hospital, Beilinson, and 4 teachers of other medical institutions. In a panel discussion, the problems of management of brittle diabetes were dealt with; furthermore, a course of 21/2 days' duration was conducted at the Government Hospital in Haifa on diseases of the liver, with the participation of a biochemist of the Medical School, in addition to the staff of the hospital.

In general, there is a struggle with the same problems as those indicated in the comprehensive study on postgraduate education and Hospitals of American Medical Association, and an endeavour to find the most suitable method adapted to the specific conditions and requirements of Israel. It is a well known fact that postgraduate studies must be geographically decentralized, and that the large hospitals in the country will become the natural centres of medical postgraduate education for the physicians in their vicinity. Owing to economic conditions, only very few physicians are able to leave their work for refresher courses of several weeks' duration, and the courses must therefore be arranged during a few days or evenings or over week-ends. Ward-rounds, case presentations, clinical-pathological conferences, x-ray conferences, panel discussions in the regional hospitals must form the central points of continuous postgraduate education for the general practitioner. One of the important questions is to find ways and means of inducing the general practitioner to take an active share in the discussion of the day-to-day problems he encounters in his work, such as minor ailments, psychosomatic disorders, early diagnosis of malignant tumors, etc.

Medical Publications and Libraries

(1) The Medical Associations of Israel publishes fortnightly "Harefuan", in Hebrew with summaries in English and French.

(2) The General Workers' Sick Fund (Kupat Holim) publishes a medical quarterly "Dapim Refuim"

(3) There exists in the framework of the National and University Library special Medical Library, comprising ca. 1400 items of medical journals and a large reading room.

(4) Government and Kupat Holim Hospitals, etc., have libraries of their own, some of them with items of considerable value.

(5) The Branches of the Medical Association of Israel have special libraries, donated by the Jarcho Fund (USA) and partly maintained still at present by this Fund, (under the direction of the National and University Library).

(6) The physicians of the Kupat Holim (ca. 1300) apart from the regional libraries receive a special subvention for the acquisition of medical books and for subscriptions to medical journals, so that almost every physician may subscribe to *Journal of the American Medical Association, Lancet,* etc.

NOTES AND NEWS

World Medical Association

MEDICAL EDUCATION CONFERENCE—The Second World Conference on Medical Education will be held in Chicago, Illinois (U.S.A.) August 30—September 4, 1959. Officers of the conference will be : President—Dr. Raymond B. Allen, Chancellor, University of California in Los Angeles, California; Deputy President—Dr. Victor Johnson, Director, Resident Training Program, Mayo Clinic, Rochester, Minnesota. In addition, the Council co-opted Dr. E. L. Turner, Secretary, Council on Medical Education and Hospitals of the American Medical Association, as a member of the Council Committee on Medical Education.

MEDICAL ETHICS AND MEDICAL LAW—The 26th Council Session of the World Medical Association adopted two principles relative to medical ethics and medical law as a result of its own efforts in a joint committee studying the subject of international medical law, and the proposed organization of an International Organization on Medical Ethics and Medical Law with national chapters. This new organization would be composed of sociologists, jurists and philosophers in addition to doctors and its aim is to codify the principles of medical ethics.

These principles are : I. The same ethical code must govern the doctor in both peace and war. II. It is the function of the World Medical Association to formulate any Code of International Medical Law and not the function of laymen even though they be lawyers.

The Council is of the opinion that The World Medical Association should be unalterably opposed to the attempts of outside groups entering a field in which they are not competent.

SECRETARIAT LIAISON CONFERENCE—Secretaries and officials of the National Member Medical Associations and the Editors of their Journals are invited to a meeting at The World Medical Association Secretariat, New York City, October 19-20, 1956. The program will be planned to facilitate mutual assistance between the Secretariats of the National Medical Associations and the Secretariat of The World Medical Association. The meeting is scheduled to follow immediately the adjourning of the 10th General Assembly and the 28th Council Session of The World Medical Association in Havana, Cuba. (10th General Assembly, October 9-15, 1956; 28th Council Session, October 16-17, 1956.)

The Secretariat Liaison Conference is the outgrowth of a meeting held at The World Medical Association Secretariat in New York City, June 1955, for National Medical Association Secretaries. The orientation and discussion proved to be successful and those who attended the first meeting were of the opinion that it should include additional officers and officials of the member associations and that such conferences should be convened whenever convenient for these people to meet at the New York Secretariat.

WORLD MEDICAL EDITORS' MEETING—The Seventh World Medical Editors' Meeting, sponsored by The World Medical Association in connection with its 10th General Assembly in Havana, Cuba, will be held on Thursday morning, October 11th, 1956 under the chairmanship of Dr. Saturnino Picaza Del Pino. The subject for discussion will be Pan American Medical Publication Problems (1. Latin American Problems in Editing Medical Journals—Dr. Augustin Delacroix, h., *Argentina;* 2. The Scientific Medical Article and Medical Ethics—Dr. Rafael Lopez Ruiz, *Colombia;* 3. The Pan American Confederation Journal as a Means of Medico-social Advancement in Latin America—Dr. Leopoldo E. Aranjo, *Cuba*). The Editor, Journal of the Indian Medical Association has been invited to attend or send a representative to the conference.

Postgraduate Courses of Instruction for Doctors

The Royal Institute of Public Health and Hygiene conducts recognised Courses of Instruction (for post-graduate medical men and women only) for the above examinations for the Conjoint Board of the Royal College of Physicians of London and the Royal College of Surgeons of England, and for the Diploma in Industrial Health examination of the Society of Apothecaries of London. The next Courses of Instruction will commence on the 28th September, 1956. Further information, entry forms and prospectuses may be obtained from the Secretary of the Institute, 28, Portland Place, London, W. 1.

Calcutta University to have College of Medicine

Calcutta University Senate approved rules on 2 June 1956 to govern the setting up of a University College for Medicine for post-graduate education in different subjects. The scheme envisages that post-graduate departments of existing institutions will co-operate so as virtually to constitute the University College of Medicine.

Contributory Health Service Scheme in Delhi

The rapid increase in the attendance of patients at the dispensaries run under the Contributory Health Service Scheme bears testimony to the growing popularity and usefulness of this pilot experiment, says the first Annual Report on the Scheme, published by the Union Health Ministry. The report reveals that while the daily average attendance at the dispensaries in Delhi and New Delhi during the first month of the introduction of the scheme in July 1954 was 3,936, it rose to 8,292 in the last week of July, 1955. The number of beneficiaries, which include Government servants and their families and dependants also increased from 2,23,000 to 2,73,844 during the same period. A significant feature of the scheme is that it has done away with the distinction between class IV and other classes of Government servants in the matter of free medical attendance and treatment.

A compulsory monthly contribution on a graded scale ranging from annas 8 to Rs. 12/- is levied on all Government employees registered under the scheme in order to ensure improved medical service.

Children's Wing of Bombay Hospital

The Governor of Bombay, Dr. Harekrushna Mahtab, declared open Mr. Hargovandas Jamnadas Ramji and Mr. Purshottamdas Jamnadas Ramji Children's Department of Sir Hurkisondas Nurrotumdas Hospital at a function held at Vanita Vishram Garden, Sandhurst Road, Bombay, on 26-5-56.

The Governor, in his presidential speech, stated that the Hospital was one of a few institutions which were being administered without any government or municipal aid. He was glad that it had rendered yeoman service to the people in the field of health.

Assam's New Public Health Plan

The Government of India, the World Health Organization and the UN Children's Fund have entered into an agreement to carry out a comprehensive public health programme in the State of Assam under an integrated plan combining elements of rural health, nursing education and maternal and child health.

Twenty-three civil hospitals and 15 maternal and child health centres formed into ten groups will participate in the scheme along with the smaller health units in Parganas or Thanas throughout the State.

The objectives of the plan include expansion and up-grading of rural health services with particular reference to maternal and child health; expansion of *dais'* training and development of training courses for sanitarians; improvement of medical and nursing undergraduate teaching and training in paediatrics; and the expansion of the present programme of health education in the State, including health education for teachers in teachers' training schools. During the project period of two years the State Government will set up 30 new health units whose function will be to provide a complete maternal and child health service, medical care, communicable disease control, sanitation, school health and health education services. Of the 480 dispensaries and 82 maternal and child health centres in various stages of development in the State now, at least 50 will be upgraded.

National Conference on Child Welfare

The first National Conference on Child Welfare was inaugurated by Shri Jawaharlal Nehru on the 9th May 1956, at New Delhi.

The Conference which was organised from 9th to 12th May, by the Indian Council for Child Welfare was attended by about 200 delegates, guest delegates, observers and visitors from the various parts of the country. More than 75 organisations were represented at the Conference.

Among the participants, there were representatives of the Union and State Governments, Community Projects Administration, Central Social Welfare Board, State Social Welfare Advisory Boards and voluntary international and national agencies. Representatives of the intra-governmental organizations, like UNICEF, WHO, FAO, UN Information Centre, etc., also attended Conference.

In the inaugural address to the National Conference on Child Welfare, the Prime Minister raised certain interesting points. He urged that child welfare schemes in India should receive priority in the national development plans and that the work had to be done in rural areas by workers having knowledge and understanding of the local conditions.

Suggesting that the child welfare work should be undertaken by the Community Project Administration and National Extension Services, which have already reached one and a half lakh of villages, Shri Nehru emphasised, "if we do not look after the children to-day, we will be creating many more new problems for ourselves in the future". He also stated that the people did not quite understand what was meant by a charter for child welfare and urged that a simple and brief charter for child welfare work in the villages be formulated. The charter should be practical and realistic and should be couched in simple language intelligible to school teachers and workers employed in the Community Projects.

Leprosy Training Course

The next Leprosy Training Course under the auspices of the Hind Kusht Nivaran Sangh (Indian Leprosy Association) will be held at the School of Tropical Medicine, Calcutta from 11 July 1956 for one month.

The course is open to all registered medical practitioners. No registration fee will be charged from candidates attending the course.

Leprosy Hospital

The Health Minister of Bombay, Mr. Shantilal Shah, formally handed over at a function held on 26 May 1956 the administration of the Kondwa Leprosy Hospital to the Poona District Leprosy Committee.

Mr. Shah said he was glad that public-spirited men who formed the Leprosy Committee had come forward to run the hospital. He was of the opinion that social workers with a missionary zeal could conduct such activities more efficiently than the Government.

Dr. N. J. Bandorewaka gave a donation of Rs. 4,000 to the hospital.

West Bengal Hospitals to be Equipped with Blood Banks

All district and sub-divisional hospitals in West Bengal are proposed to be equipped with their own self-sufficient blood banks to end their present dependence on the only blood bank in the State, located in Calcutta, for the supply of the same. These banks will be fed with the donations received from the people in their respective areas and will obviate the difficulties experienced in sending blood from Calcutta to these hospitals, particularly at the time of emergency.

As a first step to the expansion of the banks, three units will be opened at Coochehar Sadar Hospital, Berhampore Sadar Hospital and Kanchrapara Tuberculosis Hospital. The consumption of blood in these hospitals is fairly high and therefore exclusive units for them are needed.

Tuberculosis Hospital in West Bengal

A 800-bed new hospital for tuberculous patients—the third to be run by the West Bengal Government—will be ready early next year.

The site for the hospital is being selected near Dhubulia in Nadia District and about 150 acres of land are being acquired for the purpose.

The two other hospitals run by the State Government are Kanchrapara T. B. Hospital accommodating about 875 patients, in Nadia District and the M. R. Bangur Hospital at Digri in Midnapore District having about 200 beds.

All-India Obstetric and Gynaecological Congress

The 9th All-India Obstetric & Gynaecological Congress will be held in Amritsar on 29-31 December 1956. The main subject for discussion will be :—(1) Cardiac disease complicating pregnancy. (2) Malignancies of the body of the uterus. (3) Statistical survey of (i) Eclampsia and (ii) Menarche and Menopause.

The Obstetric and Gynaecological Societies all over India, as well as the teaching institutions and practitioners in the profession are invited to take part in the deliberations of the Conference.

Those desirous of reading papers on any of the abovementioned main subjects or any other subjects, are requested to communicate the title of their paper by the 1st of September, 1956 and submit typed copy of the paper in duplicate with an abstract of the same also in duplicate by the 7th of October, 1956, to Hony. Secretary, Obstetric & Gynaecological Society of Northern India, Benjamin Road, Ludhiana.

CORRESPONDENCE

The Editor is not responsible for the views expressed by correspondents

Sewage Discharge into the Jumna Causes Jaundice Epidemic in Delhi

SIR,—The daily press (*Statesman*, 1956) gives observations on the report of a Special Committee (presided over by a High Court Judge). They include :

"The Committee was, however, of the opinion that virus contamination could not have been anticipated since such a case of contamination through water supply was the first of its kind in the world".

The opinion runs counter to well-known facts :

(1) 'The virus of infectious hepatitis is present in the faeces and filtrates of faecal material from the stools of patients when fed to human volunteers have caused infectious hepatitis in from seventeen to thirty days' (Paul, 1952).

(2) 'Transmission by contacts is recognised but evidence has also been presented that water and food may be the source of infection .... poor sanitation is related to presence of the disease' (Burrows, 1949).

(3) 'Contaminated water supplies which have been shown to be one of the sources of infection, probably require special disinfection to prevent outbreaks of the disease' (Cecil and Loeb, 1951).

Incidentally, is it not time that gamma globulin, effective in preventing the disease, were prepared in the blood banks of India? A suggestion to that effect was made several years ago (Greval, 1951). The preparation was said to have been flown from America after the peak of the disease had been passed.

I am etc.,

166, Lower Circular Road,
Calcutta. S. D. S. GREVAL.

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CECIL, R. L. AND LOEB, R. F.—A Textbook of Medicine, 1951, W. B. Saunders Company, Philadelphia and London.

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PAUL, H.—The Control of Communicable Diseases, 1952. Harvey & Blythe, London.

Congenital Syphilis

SIR,—Although we are least inclined to prolong the correspondence on the Refresher article on "Congenital Syphilis", we feel that a rejoinder is called for in answer to the reply dated 16th April 1956 of Dr. Bhattacharjee to our comments dated 16th January 1956. We are sorry to find that he is quoting almost exclusively from textbooks of pediatrics and does not give or attempt to give an idea of any information based on his individual experience. He seems to be completely oblivious of the recent literature on the diagnosis, prognosis and treatment of congenital syphilis which would be found either in monographs on congenital syphilis or in journals dealing with this particular disease. Our objection to the manner and matter of his presentation of the subject intended for an average general practitioner is fully justified by the way in which he has tried to attempt and meet our comments.

The author admits that the term congenital syphilis is widely used and we may add universally recognised by the general practitioner. Since the author's article on Congenital Syphilis is primarily intended for the benefit of the general practitioners why use the term "innate" syphilis with which they are not familiar, although they mean the same thing! We are surprised that the author should quote two German authors who are sup-

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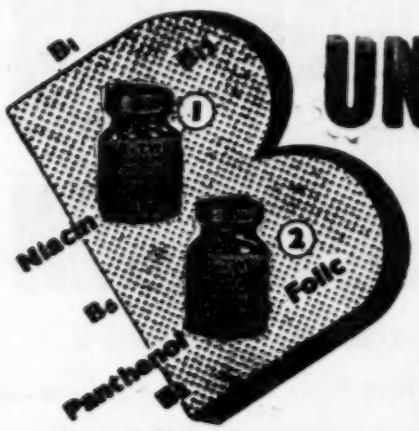

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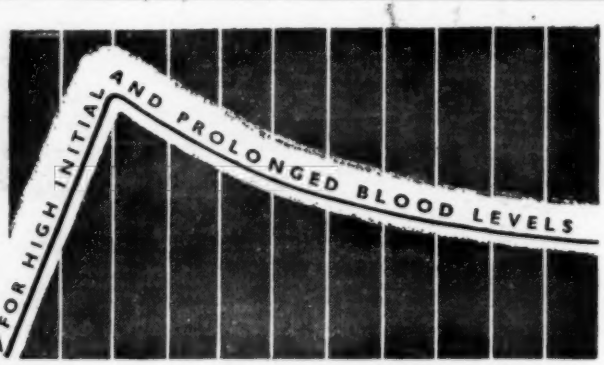

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posed to dislike the term 'congenital' as in their opinion it gives the impression that the disease has something to do with copulation. If these authors have been quoted correctly we can only feel sorry that their understanding of the English term congenital derived from the Latin word "congenitus" seems grossly defective.

According to the Oxford English dictionary reprinted in 1950, the term congenital is an adaptation from Latin *congenitus*, etymologically formed of two words *con* and *genitus* meaning born or produced along with and existing or dating from one's birth. By no stretch of etymological ingenuity the term congenital can have any connection with copulation which is derived from the Latin word *copulationem* which is translated in English as the act of coupling or the condition of being coupled and further extended to the union of the sexes in the act of copulation. None of the English or American authors whom your correspondent is quoting such as Mitchell Nelson's Textbook of Pediatrics, Brennemanne's practice of pediatrics, Sheldon's diseases of infancy and childhood, Ellis' Diseases in infancy and childhood have used the term "innate" for congenital. The uncorroborated, unconfirmed report of the existence of spirochaetal nests in latent congenital syphilis by Muller as quoted by the correspondent is unacceptable as no pathologist of repute in the English speaking countries has described any such condition. The correspondent has quoted Mitchell Nelson's Textbook of Pediatrics for his statement that secondary pyogenic infection may occur as a complication in syphilitic metaphysitis and has drawn our attention to the photograph in the book. While thankful for the reference, we tried to find corroboration to that statement from other books on pediatrics and authoritative books on syphilis such as Stokes, Jeans and Cook and books on pathology and fail to find that suppuration of syphilitic metaphysitis is a common occurrence. It is theoretically possible for a child with congenital syphilis to suffer from bacteraemia and sustain a pyogenic osteomyelitis which may even be superimposed on an already existing syphilitic metaphysitis. In a series of 893 cases of early infantile congenital syphilis which the authors have examined and treated over a period of 25 years, 75 to 85 per cent of these infants showed roentgenological evidence of osteochondritis, periostitis etc., with or without lesions of skin or mucous membrane. They have not come across a single case of suppuration and sinus formation in the bones already affected by syphilis. The authors are unable to accept the statement of Mitchell Nelson that suppuration of the bones and soft tissues due to secondary invasion by pyogenic organisms through the medium of the blood stream is *not an uncommon finding in children with congenital syphilis*. It is theoretically possible that a bacteraemia with pyogenic organisms or even tubercle bacilli may occur in a congenital syphilitic infant, but it is excessively rare. It is a pity that your correspondent has been quoting exclusively material from textbooks on pediatrics and has not cared to refer standard books on syphilology—both the clinical and pathological aspects of congenital syphilis.

With regard to the involvement of central nervous system in congenital syphilis we are glad that your correspondent's statement in his original article that changes in the C.S.F. are seen in more than 75 per cent of cases has now been corrected to 35 per cent which means a considerable difference. Here again he has taken the trouble to quote eminent authors like Nabarro etc., whose figures on percentage involvement of central nervous system may be true so far as children in those countries are concerned. The author should have given some indication whether such high incidence of involvement of central nervous system in congenital syphilis obtains in our country. For the information of the author of the refresher article who quotes Katherin Dodd for the statement that "neuro-syphilis is unfortunately the second most common lesion of late congenital syphilis", we are certain that the statement does not apply to the incidence of neuro-syphilis in our country, and during a 25 year period we have come across only *3 cases of neuro-syphilis among 178 late congenital syphilitics*, while interstitial keratitis and tertiary lesions of the skin and bone were the commonest clinical conditions met with in hospital practice.

On the question of treatment of congenital syphilis it is unfortunate that the correspondent should quote Willcox's survey and give only a minority figure of 28·9 per cent of the clinics using penicillin in conjunction with other drugs, while he has completely omitted to state that as per survey by the same author 91 per cent of clinics in America and Asia are using penicillin alone in the treatment of syphilis. The Expert Committee on Venereal Diseases and Treponematoses of the WHO reviewed the whole position of treatment of syphilis and have only recommended the use of penicillin alone in the treatment of early acquired syphilis, prevention of congenital syphilis and the treatment of early infantile syphilis based upon the organised long range studies of countries over a period of 10 years. It is rather too late in the day for the correspondent to quote the outmoded use of a toxic drug like acetarsan in the treatment of infantile congenital syphilis. Leaving aside the use of penicillin, acetarsan, a pentavalent arsenical compound has been found to be therapeutically distinctly inferior to the trivalent arsenicals such as neoarsphenamine and sulpharsphenamine and was found to be much more toxic than the later. Further, treatment with arsenicals whether oral or parenteral requires multiple courses spread out over a period of 12 to 18 months for cure. Does the correspondent seriously think that he would recommend this course to the general practitioners in preference to penicillin with which the treatment is completed in a period of 8 days? It is extraordinary that he should recommend treatment with acetarsan etc., in which the co-operation of the parents in administering the drug regularly by mouth for a longer period is an unknown and undependable factor. The authors wish that the Editor of the journal could have published the standard, recognised schedule of treatment with penicillin which we have indicated in our first article.

We are etc.,

Institute of Venereology,
Govt. General Hospital,
Madras.

R. V. RAJAM,
P. N. RANGIAH.

REVIEWS

Pediatric X-ray Diagnosis—By John Caffey, Third edition, 1956. The Year Book Publishers, Inc., Chicago, 18 × 25 cm., pp. xxiv + 1059, price $28.

Without going into the technique of radiography the author has attempted to be of practical help to paediatricians and radiologists by incorporating in the book a wealth of material. The book is up-to-date and many controversial issues are clinched while the common findings are not overlooked. Cooley's anaemia, apart from blood changes, can radiologically manifest only in the extremities instead of the skull while typical radial striations of the skull may be found in spheroidal anaemia (familial haemolytic icterus) and also in sickle shaped anaemia. This is well illustrated with pictures. A case of infantile cortical hyperostosis of clavicle and ribs as distinct from dyschondroplasia of Albers-Schönberg type is presented. By introducing contrast media into the amniotic fluid it has been shown that respiration is established while the foetus is within the uterus. Reversibility of cylindrical bronchiectasis in atelectasis lung, postulated by Findlay is demonstrated with skiagrams. Pathogenesis of tuberculous affection of the lungs has been well summarised and has been helpful in comprehension and interpretation of radiological findings. In some cases corroboration by autopsy of the findings in skiagrams has been convincing. While appearance of the lungs in conditions of atelectasis of its different portions is shown in different skiagrams there is no pointer how to distinguish a localised atelectasis of the lung adjoining the superior mediastinum from other causes of enlarged mediastinal shadow. The importance of anterior projection with the patient lying on his side in cases of hydro- or pyopneumothorax is not mentioned. Presentation of the various normal irregularities of the pelvic bone and of the small bones of the hand and foot should be a warning against enthusiastic pathological diagnosis. Rare diseases like Ehlers-Danlos syndrome showing calcification of subcutaneous tissue or Ellis Crevald syndrome, a special variety of chondrodystrophy are also illustrated. The style is lucid and reproductions of skiagrams excellent.

Management of Pain in Cancer—Edited by M. J. Schiffrin, PH.D., Foreword by Warren H. Cole, M.D. Year Book Publishers, Inc., Chicago, U.S.A., 1956. 18 × 13 cm., pp. 245. Price $4.50 post paid.

This is a neat little book dealing with a subject which, though important to the patient and his doctor, seems to have received insufficient attention in medical literature. Every year a large number of patients, with cancer in the terminal stages, pose very serious practical and exacting problems—to themselves, to their near and dear ones, and also to the physicians looking after the unfortunate sufferers. Of these multifarious problems, the relief of pain is indeed a difficult, significant and a very vital one; for, the severe and constant pain commonly seen in the last stages of cancer destroys the stamina and morale of even the strongest individual and is heart-rending for the onlookers. This book, contributed by eight authors, aims at guiding the family physician to help mitigate the patient's sufferings. Some of the chapters are directly useful to him, some others help him to achieve technical competence for various procedures, while others again help him to understand and offer better co-operation to specialists and experts who might be treating the patient.

Preparing for Motherhood, a Manual for Expectant Parents—By Samuel R. Meaker, M.D. Published 1956 by the Year Book Publishers, Inc., Chicago, U.S.A., Board-bound, pp. 196; price $2.00.

The author of this book has had extensive experience in obstetric and gynaecological practice and is Professor Emeritus of Gynaecology at the Boston University School of Medicine, where he has taught for more than thirty years. The subject has been discussed in a personal friendly way in 17 convenient chapters, all of which have been enriched by drawing upon the wide experience of the author. The treatment of the subject on the whole makes this book appear somewhat different from several similar books which have passed through the hands of this reviewer. One interesting and important deviation is that the author takes also the expectant father into consideration and confidence—thereby ensuring a happy and co-operating team of the couple into preparing for and welcoming the new arrival. The style and language are clear, explicit and easily understood. Although naturally the author addresses the expectant parents with the background of conditions and practices prevalent in the U.S.A., the directives, advice and instructions hold good universally—except perhaps in minor details here and there. Slightly bigger types would have made reading this very useful book more comfortable.

Genetics in the Atomic Age—By Charlotte Auerbach, PH.D., D.SC. Published 1956 by Oliver and Boyd, pp. 106; price sh. 8/6 net.

The subject of genetics does not seem to have created much interest and enthusiasm amongst the educated public outside the fold of the geneticists themselves. This may perhaps partly be due to not many books being available for "public" consumption, written in non-technical terms. The volume under review is an excellent book from this point of view, written as it is in simple, easily-understood and non-technical language. Dr. Auerbach has treated the subject in a very interesting way, tending to stimulate further thought and reading. The basic principles of genetics, reproduction and heredity are first described; then the reader is introduced to the subject of mutation—normal or spontaneous and then artificial, under extraneous influences like radiation, atomic explosions and the like with discussions of their possible hazards to posterity from the genetic point of view. Much credit is due to Miss I. G. Auerbach for the illustrations in keeping up the interest in going through this very readable book.

SUPPLEMENT
Journal of the Indian Medical Association

VOL. 27, No. 1 CALCUTTA JULY 1, 1956

BRANCH NOTES

ADILABAD BRANCH—A meeting of the branch was held on 10-2-56. Office bearers for 1956-57 were elected with Dr. M. Mousikuddin as president, Dr. D. A. Mohan Rao as hony. secretary and Dr. Miss Amiat Unnisa Begum as hony. joint secretary.

ALIGARH BRANCH—A meeting of the branch was held on 18-12-55 with Dr. D. P. Sharma in the chair. The proceedings of the last meeting were confirmed. The appeal of Dr. S. C. Sen, president for collection of donations towards the Building Fund was discussed and members were requested to raise its proportionate share as early as possible.

* * * * *

A meeting of the branch was held on 22-1-56 at the village of Kasimpur. To hold meetings in village was a new experiment which was appreciated by all members. Thirty members and 4 guests were present. The members decided to contribute Rs. 10/- each before March 1956. Members were informed of the Study Tour in Europe arranged by IMA in collaboration with Air India International. A rare case of Sensory Disturbances involving only thermal sense and patchy absence of sweating was presented by Dr. Jolly.

ALLAHABAD BRANCH—A meeting of the branch was held on 26-1-56 and the members condoled the death of Dr. Bhawa Nath Jha, a founder member of the association.

* * * * *

A meeting of the branch was held on 21-1-56. Dr. J. C. Mehta, I.M.O. Northern Railway, Allahabad, spoke on Jaundice. Dr. S. N. Chatterjee, Civil Surgeon, demonstrated two cases of foreign body in stomach.

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A meeting of the branch was held on 15-2-56. Dr. J. C. Mehta demonstrated 4 interesting cases. Dr. S. N. Chatterjee discussed one case of gun-shot wound.

* * * * *

A meeting was held on 3-3-56. Dr. A. K. Ghosh read a paper on Common Laboratory Test.

ALWAR BRANCH—A clinical meeting of the branch in which members from the district towns also participated, was held on 18-2-56, with Dr. C. M. Sharma in the chair. Twenty-two members were present. Dr. B. M. Kothari gave an interesting talk on "Infective Hepatitis". Dr. P. N. Mathur described 3 very interesting cases he came across in his medical career. Dr. C. M. Sharma invited the younger members to participate in discussions.

ARARIA BRANCH—Health Week was celebrated under the auspices of this branch, from 12-18 March 1956. Lectures on general health and hygiene, vaccination, disinfection, mass inoculation were given. Baby shows were arranged and prizes given.

ASANSOL BRANCH—A clinical meeting of the branch was held on 18-12-55. Dr. B. C. Sinha, a member of the International Society of Cardiology, delivered a lecture on Coronary Thrombosis and its management.

BAHRAICH BRANCH—A meeting of the branch was held on 12-2-56. Seven members were present. Dr. B. N. Chatterjee presided. Dr. D. N. Mukherji was elected secretary of the branch in place of Dr. Agarwal on transfer. Dr. M. N. Srivastawa demonstrated 3 cases of (1) a typical Appendicitis, (2) Bicornuate Uterus and (3) Chronic Bronchitis with Congestive Heart failure.

* * * * *

A meeting was held on 5-3-56. Dr. B. N. Chatterjee presided. Ten members were present. The members condoled the death of Dr. M. N. Saha, Sri G. V. Mavlankar and Acharya Narendra Dev.

* * * * *

A meeting was held on 12-4-56. Dr. B. N. Chatterjee presided. Six members were present. A sub-committee for the II Five Year Plan was formed to help the District Planning Committee in medical and health matters. Dr. D. N. Mukerjee spoke on D.D.T., demonstrated cases of Rheumatic Endocarditis, Tubercular Adenitis and T. B. Spine. Dr. D. N. Mukerjee presented a case of Malignant Neutropenia.

BANARAS BRANCH—A meeting of the branch which was held on 25-2-56, condoled the sudden death of Dr. C. M. Bose, one of the old members of the association.

BANGALORE BRANCH—On 17-12-55, a meeting of the Managing Committee was held under the presidentship of Dr. P. Krishna Rao. Dr. B. Hanumaiah gave a lecture on "Bleeding per Anus". Dr. T. Seshachalam presided.

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On 31-12-55 Dr. Jivaraj Mehta, Finance Minister of Bombay, addressed the members of the Association.

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On 17-1-56 Dr. M. N. Gupta, Dy. Adviser for Factories, Government of India, gave a lecture on "Health Hazard in Industries". Dr. M. Sivaram presided.

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On 24-1-56 Prof. C. S. Powell spoke on "The Relationship of Physics to Medicine". Dr. C. V. Natarajan presided.

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On 11-2-56 Dr. C. B. Leibman spoke on "The Use of Isotopes in Medicine". Dr. M. Sirsi presided.

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On 20-2-56 Dr. Rose Goldon, Visiting Professor of Radiology, Medical School, University of California, spoke on "Some Aspects of Gastro-enterology" (with demonstrations of slides). Dr. B. Venkatasubba Rao, Director of Medical Services in Mysore presided.

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A meeting of the Managing Committee of the branch was held on 16-4-56 with Dr. P. Krishna Rao in the chair. A symposium on Infections of the Hand was held. Drs. T. S. Jairam, M. Authikeshavalu, B. Hanumaiah and T. Seshachalam participated. A film on Occupational Therapy was also shown.

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On 22-5-56 Dr. A. V. Ivy, Chairman, Medical Science, Illinois University, spoke on Peptic Ulcer. Dr. B. Venkatasubba Rao, Director of Medical Services of Mysore, presided.

BEGUSARAI BRANCH—An emergent meeting of the branch was held on 8-3-56. An elaborate programme was chalked out for the celebration of the Health Week.

BHIR BRANCH—A meeting of the branch was held on 11-3-56. Dr. G. Nabi, Civil Surgeon, presided. Eleven members were present. Dr. H. R. Kandlikar read Dr. S. N. Deshpande's paper on Tetanus, due to author's absence. Dr. K. Rashed Ahmad, M.O., Manjlegaon, spoke on "Cortisone in Tuberculosis".

BIJAPUR BRANCH—The annual general meeting of the branch was held on 6-11-55 with Dr. Mangalvedhekar in the chair. Twenty-six members were present. Office-bearers were elected with Dr. G. N. Mangalvedhekar as president and Dr. M. D. Mahindrakar as hony. secretary.

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A meeting of the branch was held on 11-12-55 with Dr. S. B. Patil in the chair. Dr. V. G. Joshi president of the M. & K. Provincial Branch was the chief guest. Dr. Joshi in his speech advised members to be co-operative among themselves.

CHANDA BRANCH—The annual general meeting of the branch was held on 4-3-56 with Dr. K. V. Chary in the chair. Office bearers for 1956-57 were elected with Dr. W. M. Mathkar as the president, Dr. K. V. Chary as hony. general secretary and Dr. T. V. Pattiwar as hony. joint secretary.

CHETTINAD BRANCH—A meeting of the branch was held on 18-2-56. Dr. Miss H. Erikson presided. Dr. Israel demonstrated 2 cases (1) Operated Case of Ankylosis Jaw and (2) Chronic Parotitis. Dr. Wallden demonstrated a case of Cerebral Palsy. Dr. Miss S. Person spoke on the Education and Occupational Therapy in cases of Cerebral Palsy. Dr. D. S. Manuel read a paper on "Extra Intestinal Amoebiasis". Dr. S. G. Sundaram presented a case of Pulmonary Amoebiasis with skiagrams.

CHITALDURG BRANCH—A meeting of the branch was held on 21-4-56 conducted by Dr. D. Shammana, the District Medical Officer. Dr. D. P. Jayaram of Sri Krishnarajendra Hospital spoke on the Management of Eczema. A hearty send off was given to Dr. B. N. Chennabasappa on the eve of his transfer. The members were requested to donate to the Building Fund.

COCHIN BRANCH—The members participated in the 6th annual provincial conference held on 15-16 October 1955.

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A meeting was held on 9-12-55. Dr. M. Krishnan Nambissan spoke on "Some Aspects of Tuberculosis".

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An emergent meeting was held on 15-12-55 to consider the government proceedings relating to the pay scales of the medical officers of the State and to offer our recommendations.

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A meeting was held on 24-1-56. Dr. N. Vasudevan spoke on Modern Trends in Medicine.

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A meeting was held on 14-2-56. Dr. S. Sacharias spoke on Collagen Diseases.

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An emergent meeting was held on 28-2-56 to condole the death of Dr. Jacob Taliah.

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An emergent meeting was held on 11-3-56 to condole the death of Dr. K. Achutha Marav of Ophthalmic Hospital, Ernakulam.

COIMBATORE BRANCH—A meeting of the branch was held on 21-4-56. Dr. D. Sundareswaran presided. Sixty-five members were present. The meeting condoled the death of Dr. J. A. S. Masilamani of Government General Hospital, Madras and of Dr. R. E. S. Mathiah of Government Ophthalmic Hospital, Egmore. Dr. R. Kalamegham of Trichy spoke on "The Limping Child".

DALTONGANJ BRANCH—A meeting of the branch was held on 22-2-56. Seventeen members were present. D. R. Sinha was elected president in place of Dr. S. A. Muzaffar, Civil Surgeon retired. The meeting condoled the death of Dr. R. Dayal, ex-president of the branch and Dr. Rajeswar Prasad of Arrah. An appeal was made to all members to donate to the State Building Fund. Dr. Sinha spoke on the "Role of Antibiotics and Chemo-therapeutics on the Diseases of the Eye". It was decided to secure a piece of land for a building of the branch.

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Health Week was celebrated from 12-18 March 1956, under the auspices of this Branch. The notable features of this year's celebration were mass inoculation, vaccinations and disinfection of wells in the rural areas as well. The public were enlightened on the modern conceptions of the prevention of communicable diseases and health pamphlets were freely distributed all over the district.

A large number of prizes were awarded for baby show, sanitation drive, good physique, modern sanitary and economic houses at Garhwa, Japla, Pandi Latehar and Daltonganj.

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A meeting of the branch was held on 30-4-56. Dr. R. Sinha, Civil Surgeon, Palamau, presided. Fifteen members were present. Dr. G. S. Pathak of Sadar Hospital, Daltonganj, was given a farewell on his transfer to Motihari.

DEORIA BRANCH—A general meeting of the branch was held on 12-2-56 with Dr. (Mrs.) S. Mohindra in the chair. Fourteen members including 2 new members were present. The symposium on Typhoid was restarted by Dr. B. P. Mohindra. Dr. S. C. Acharya also took part in it.

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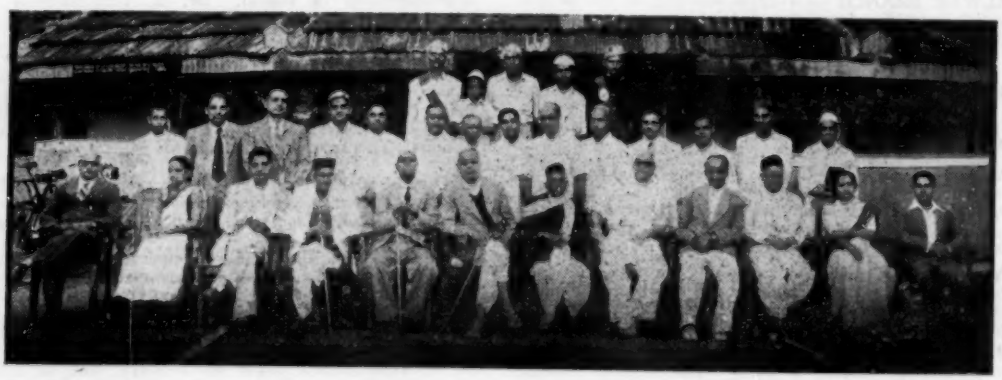
A general meeting of the branch was held on 6-5-56. Dr. (Mrs.) S. Mohindra presided. Seven members were present. The meeting acceded to the request of the



MEMBERS OF THE BIJAPUR BRANCH ASSEMBLED AT THE ANNUAL GENERAL MEETING OF THE BRANCH HELD ON 6-11-55.

District Magistrate to have the services of the members when necessary during the Buddha Jayanti Celebrations. It was proposed that the branch should construct a building of its own. Dr. Jagatnarain promised a contribution of Rs. 100/- only. Dr. S. C. Acharya read a paper on Leishmaniasis.

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A general meeting of the branch was held on 4-3-56. Dr. S. N. Mukherji presided. Seven members were present. After the routine business was over, the Memorandum, Rules and Bye-Laws of the branch as framed by Dr. S. C. Acharya were passed with modifications. Dr. S. C. Acharya read a paper on Amoebiasis. Dr. R. M. Singh cited a case of Tubercular Meningitis, Dr. N. C. Jain a case of Tubercular Endophthalmitis.

DHARWAR BRANCH—The annual general meeting of the branch was held on 12-2-56. Office bearers for 1955-56 were elected with Dr. W. H. Gokhale as president and Dr. H. V. Upadhye as hony. secretary.

DIGRI BRANCH—The first clinical meeting of the branch was held on 22-4-56 with Dr. M. M. Roy in the chair. Dr. S. K. Ganguli introduced a discussion on "Recent Trends of Chemotherapeutic Treatment in Pulmonary Tuberculosis and Minor Collapse Therapy and Postural Recumbency in Domiciliary Regime". More than 60 doctors from Jhargram, Tamluk, Garbeta, Midnapore, Kharagpur and other neighbouring places attended the meeting.

FEROZEPORE BRANCH—An extraordinary meeting of the branch was held on 29-4-56. Dr. Rajaram Bhola presided. Fourteen members were present. Five members of the Provincial Medical Council were also present. Dr. Kapur pleaded for membership drive.

GARHBETA BRANCH—The 10th Anniversary meeting of the Indian Medical Association, Garhbeta Branch was held at Kadra on 4-3-56; in the morning a socio-medical conference was held at Garhbeta Cinema Hall inaugurated by Dr. R. C. Adhicary and presided over by Dr. N. K. Munshi, president, Bengal Provincial Branch. Dr. K. P. De, hony. secretary, Garhbeta Branch and vice-president, Bengal Provincial Branch said this type of conferences would help to awaken health consciousness among the people and to establish better understanding between the profession and the public. Dr. Adhicary, Dr. Munshi, Dr. J. Majumdar, Dr. L. K. Ganguli and some members of the local profession spoke about prevention and relief of diseases.

Then the distinguished guests from Calcutta and others took part in the Rural Medical Conference at Kodra. About 40 doctors were present. Dr. N. K. Munshi presided. The Conference discussed the problem of dispersal of medical personnel, quackery, school health, relation with the public and public bodies, remuneration in the rural areas and dissociation from ayurvedic and homeopathic institutions.

GAYA BRANCH—The annual general meeting of the branch was held on 17 October 1955 in the newly erected building of the association. Dr. R. Sinha presided. The annual report and accounts of the previous year were passed. Office bearers for the next year were elected with Dr. J. N. Mehrotra as president, Dr. S. N. Chakerburty as hony. general secretary and Dr. S. K. Mitra and Dr. G. P. Keshri as hony. joint secretaries.

GHAZIPUR BRANCH—A meeting of the branch was held on 8-4-56. The members formed a sub-committee to consider the working of the II Five Year Plan. Dr. S. H. Ahmed read a paper on "Hormones as Therapeutic Agents."

GURGAON BRANCH—A meeting of the branch was held on 22-4-56. Dr. Jagjit Singh presided. Nineteen members were present. It was decided to request the Government for exemption of fees for X'ray and Laboratory examinations in Government Hospitals.

JAGATDAL BRANCH—A clinical meeting was held on 13-3-56 with Dr. S. C. Bhattacharjee in the chair. Dr. S. N. Ganguly, Civil Surgeon, Hooghly, spoke on the Treatment of Heart Failure.

JAMSHEDPUR BRANCH—The annual general meeting of the branch was held on 8-4-56. Dr. D. N. Mookerji presided. Fifty-two members were present. The report of the Hony. Secretary for the year ending 31-3-56 and the statement of accounts were passed. Office bearers for 1956-57 were elected with Dr. J. K. Ghosh as president, Dr. R. C. Tarapore as hony. general secretary and Dr. S. K. Ganguli as hony. joint secretary.

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On 12-4-56, the foundation stone of the association building was laid by Lady Ghandy before a distinguished gathering.

DR. A. C. UKIL, DR. S. C. SEN AND DR. C. M. MEHTA WITH THE EXECUTIVES OF THE GUJARAT AND SAURASHTRA PROVINCIAL BRANCH, AHMEDABAD ON 4-3-56.

KALYANI BRANCH—All India B.C.G. Day was observed on 20-2-56 at the Kanchrapara T.B. Hospital under the auspices of the branch. The president and the secretary of the branch, the superintendent and the R.M.O. of the hospital thoroughly discussed the importance of B.C.G. vaccination.

KANPUR BRANCH—The foundation Anniversary Week of the branch was celebrated from 28-2-56 to 5-3-56. On 28-2-56 a symposium on Respiratory Diseases in Children was held with Dr. B. N. Bhallay as chairman. The chief speaker was Dr. N. Subbie. On 3-3-56, a symposium on Common Manifestation of Tuberculosis and its Treatment was held, Dr. R. S. Dixit was the chairman, Dr. R. N. Tandon was the chief speaker. On 5-3-56, a symposium on Otoselerotic Deafness and its Treatment was held. Dr. C. S. D. Misra was the chairman and Dr. R. N. Misra was the chief speaker.

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A meeting of the branch was held on 11-3-56. Dr. S. P. Gupta, Professor of Ophthalmology, K. G. Medical College, gave a lecture on "Corneal Ulcer and its Treatment".

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In a meeting held on 22-4-56 the members condoled the death of Dr. S. N. Misra, an ex-president of the branch. Dr. R. Prasad presided.

KARNAL BRANCH—A clinical meeting was held on 1-4-56. Dr. Nirmal Parkash presided. A paper on "Dyspepsia" was read by Dr. Om Prakash of Pehwa. This was discussed by some of the members present. The members decided to contribute Rs. 5/- each towards the Building Fund.

The members gave a farewell party to Dr. Nirmal Parkash, the president on 11-4-56, on his transfer to Ambala.

KHAMGAON BRANCH—The half yearly report of the branch shows that membership has come up to 10. Meetings were held regularly every month.

KOTHAGUDIUM BRANCH—At the clinical meeting held on 23-11-55 Dr. I. Kuppuswamy gave a talk on "Cardiac Arrest during Anaesthesia and its management".

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At the clinical meeting held on 28-12-55, a demonstration of the working of the Cardiostat was done by Mr. P. R. Pattabiraman.

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At the usual clinical meeting held on 11-1-56, Dr. S. K. De gave a talk on "x'rays and their application in diagnostic radiology".

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On 18-1-56 in a meeting, Dr. (Mrs.) E. Kuppuswamy read a paper on "Management and Treatment of Burns according to modern technique", and Dr. S. V. Ghurye, gave a talk on "Electrocardiograms and the Interpretation of their records in various heart disease".

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Dr. T. C. Ramchandani, President of the I.M.A., Kothagudium branch, gave a lively talk on his trip to United Kingdom, at the clinical meeting held on 18-2-56.

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On 29-2-56, Dr. S. V. Ghurye, demonstrated two interesting cases of Mediastinal Tumours. He described in detail the pathology, differential diagnosis and treatment.

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During the usual clinical meeting held on 17-3-56 the following cases were demonstrated. Dr. S. V. Ghurye, demonstrated a case of Nephritis and a case of Infective Hepatitis. Dr. S. Vaidyanathan, demonstrated (1) a case of Brachial Plexus paralysis after an accident and (2) a case of Cancer Rectum. Dr. H. M. Jagannatha Rao, demonstrated a case of Tumour Abdomen.

KRISHNA DISTRICT BRANCH—A meeting of the branch was held on 14-4-56 with Dr. N. Govindarajulu in the chair. Lt.-Col. P. Papatla, Director of Medical and Public Health Departments, Orissa spoke on Common Eye Diseases in General Practice.

MADURA BRANCH—The annual general body meeting of the branch was held on 3-3-56. Office-bearers for 1956-57 were elected with Major G. A. Naidu as president and Dr. K. A. Ramlingam as secretary.

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A meeting of the branch was held on 21-4-56 with Dr. (Major) G. A. Naidu in the chair. The meeting condoled the death of Drs. J. A. S. Masilamony, Muthiah and Gopalaswami. Dr. C. S. Sadasivam spoke on some common surgical pediatric problems.

MANDAPETA BRANCH—A meeting of the branch was held on 27-2-56 with Dr. C. Narasimharao in the chair. A symposium was held on "Cough and its Management".

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A meeting of the branch was held on 23-3-56 with Dr. Ramabrahman in the chair. Dr. Francis gave a lecture on "Filariasis".

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A meeting of the branch was held on 15-5-56. Dr. C. Narasimharao presided. Dr. A. Kameswara Rao discussed a case of "Cirrhosis of the Liver" in a child of 8 years. Dr. K. Ramabrahmam presented a case of Tetanus in a child of 2 years.

MANDYA BRANCH—A meeting was held on 4-3-56 with Dr. H. Samasastry in the chair. The Mysore Medical Association was requested to hold the next conference at Mandya. Dr. A. K. Gopalarajan, Superintendent, K. R. Hospital, Mysore, spoke on "Abdominal Emergencies".

MEERUT BRANCH—A meeting of the branch was held on 14-1-56. Dr. P. C. Dhanda of Delhi spoke on "Some aspects of Respiratory Insufficiencies".

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A general meeting of the branch was held on 14-2-56. Forty doctors were present. Dr. J. N. Madan presided. The proceedings of the last meeting were confirmed. Dr. P. N. Taneja spoke on Paediatrics and the General Practitioners.

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A meeting of the branch was held on 24-3-56. Thirty-three doctors were present. The proceedings of the last meeting were confirmed. Dr. D. Comar of Delhi gave a talk on "The Role of Surgery in Cardiovascular Disease".

MONGHYR BRANCH—An emergent meeting of the branch was held on 11-5-56. The meeting condoled the death of Dr. M. N. Verma, an energetic member of the branch.

MUZAFFARPUR BRANCH—The branch celebrated the Health Week from 12-18 March. The programme included popular lectures, sanitation drive, mass inoculation, sports etc. At Panapur and at Muzaffarpur, baby shows were arranged.

PATLAD BRANCH—A meeting of the medical men of Petlad was held on 1-4-56 under the chairmanship of Dr. N. G. Desai and a branch of I.M.A. under the name of Petlad branch was opened with Dr. N. G. Desai as president and Dr. H. C. Shah as secretary.

PORBANDAR BRANCH—A meeting of the branch was held on 21-4-56. Dr. H. H. Chavda presided. Thirteen doctors were preseat. Dr. H. A. Maniar of Bombay, spoke on Common Skin Diseases.

RAMNAD BRANCH—The annual meeting of the branch was held on 25-3-56. Dr. M. D. Ananthachari, Principal of Madura Medical College presided. Office bearers for 1955-56 were elected with Dr. R. Venkatachari as president and Dr. S. Radha Krishnan as secretary. Major G. A. Naidu spoke on "Virus Diseases of the Skin" and Dr. K. S. Krishnan gave a talk on "Vaginal Discharge". There was also a film demonstration on Virus Research.

The annual report of the branch for 1954-55 shows a slight fall in membership. Six meetings were held during the year. There is noticeable improvement in finance.

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A meeting of the branch was reld on 6-5-56 with Dr. R. Venkatachari in the chair. Dr. V. Srinivasam spoke on "Therapy in Cardiac Failure".

RATNAGIRI BRANCH—At a meeting held on 19-2-56 with Dr. (Miss) C. Pannikar in the chair, the members condoled the death of Dr. B. M. Sharma, the president of the branch.

ROHTAK BRANCH—On 20-2-56, the president of the branch welcomed the president, Dr. B. L. Kapoor, and secretary, Dr. Faqir Chand Shori, of the Punjab State Branch at annual dinner. In his speech the president of the branch discussed the place of modern medicine in this country and the conduct of medical men in the practice of medicine. Strong leadership amongst the profession he said would raise the profession to its honourable positions.

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At a meeting of the branch held on 1-3-56 under the presidentship of Dr. P. R. Sondhi, the members condoled the death of Dr. Jagdish Chandra's father.

SALEM BRANCH—At the annual general body meeting held on 31-3-56, Dr. P. S. Ramaswamy was elected the president, Dr. V. Raghavendra Rao, the hony. secretary and Dr. S. Arthanarai, hony. joint secretary for the year 1955-56.

SECUNDERABAD BRANCH—A meeting of the branch was held on 19-3-56. The members condoled the death of Dr. S. S. Kirtane on 12-3-56 and of Dr. Y. N. Timmaraju on 17-3-56.

SEHARSA BRANCH—A meeting of the branch was held on 23-3-56 with Dr. S. P. Mukherjee in the chair. Dr. J. Choudhury placed before the house, the deeds (documents) of the land for the Association building. The secretary appealed for funds for the building.

SERAMPORE BRANCH—A new branch was opened at Serampore on 10-4-56 by Dr. N. K. Munshi. Dr. A. Sinha, joint secretary, I.M.A. Bengal branch, discussed the utility of the Indian Medical Association. Dr. N. Bhattacherjee and Dr. J. Mukherjee were elected the president and the secretary of the branch respectively.

SHILLONG BRANCH—A meeting of the branch was held on 20-2-56 to condole the death of Dr. U. C. Dutta, a member of the branch. Dr. D. K. Rao of the International Planned Parenthood Association spoke on Family Planning. Thirty members were present.

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A meeting of the Executive Committee of the branch was held on 7-3-56. The meeting viewed with great concern the recent and retrograde decision of the Shillong Municipal Board to abolish the Segregation Hospital. It also regretted that the Municipal Board was charging -/2/- annas per dose of vaccine lymph for vaccinating people against small pox and requested the Board to do it free.

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At a meeting of the branch held on 25-4-56, the members condoled the sad and sudden death of Dr. P. N. Ghosh of Dibrugarh who was a past president of the Assam State Branch of I.M.A.

SHIMOGA BRANCH—A clinical meeting of the branch was held on 18-2-56. Dr. M. V. Shanbhog presided. Dr. D. Krishnamurthy demonstrated 2 cases of (1) Haemoptysis due to a foreign body, (2) Vitamin A Deficiency associated with Hepatic and Cardiac enlargement. Dr. T. N. Srinivasaih spoke on Umbilical Adenoma.

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A clinical meeting of the branch was held on 7-4-56. Dr. T. I. N. Pandit, District Medical Officer, presided. Dr. M. Putttaiya of Chickmaglur spoke on "Death and the Doctor". Dr. A. S. Kantharaja Setty demonstrated a case on "Treatment of Diphtheria".

SIBSAGAR BRANCH—At a meeting held on 17-4-56, the members condoled the death of Dr. P. N. Ghose, ex-president of the Assam State Branch.

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A meeting of the branch was held on 12-5-56. Dr. B. R. Shama Rao presided. Dr. S. V. Govinda Setty of Sri Krishnarajendra Hospital, Mysore, spoke on "Pitfalls in the treatment of Diabetics".

SITAPUR BRANCH—The annual meeting of the branch was held on 4-2-56. The report of the last year (1955) and the accounts were passed. Office-bearers were elected with Dr. N. B. Misra as president, other office-bearers of the last year were re-elected.

TENALI BRANCH—A meeting was held on 19-11-55 with Dr. V. Sundararamaiah in the chair. Dr. P. Venkatarao of General Hospital, Guntur, spoke on "Recent Advances in Anaesthesia" including controlled Respiration and controlled Hypotension.

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A meeting was held on 21-1-56 with Dr. V. Sundararamaiah in the chair. Dr. V. Surjanarayana spoke on "Surgical Emergencies in Ophthalmic Practice".

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A meeting was held on 26-2-56 with Dr. V. Sundararamaih in the chair. A symposium on "Anaemias" was held, Dr. C. Sitaramaiah of Guntur Medical College spoke on "Physiology of Haemopoiesis"; Dr. P. Lakshmanrao of General Hospital discussed the classification of Anaemias; Dr. P. Narayanrao of Guntur Medical College dealt with the Evaluation of Anti-Anaemic Drugs in the treatment of anaemias. Dr. C. Krishnaiah spoke about the "Surgical Aspects of Anaemia" and lastly Dr. A. Vasantharao spoke on "Blood Transfusion in Anaemia".

TIRUCHY BRANCH—A meeting of the branch was held on 4-2-56. Dr. V. Enok presided. Sixty members were present. Dr. N. S. Narasimha Iyer give a talk on Treatment of Fractures in outlying stations.

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A meeting of the branch was held on 18-2-56 with Dr. V. Enok in the chair. Fifty members were present. Dr. K. C. Nambiar spoke on Role of General Practitioner in the Field of Surgery. Dr. Indra Ramamurthy spoke on a few advances in Obstetrics and Gynaecology.

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A meeting of branch was held on 25-2-56. Twenty-five members were present. Dr. A. Umapathy Mudaliar, Medical Officer, Tiruchy, presided. Dr. P. Vadmolyan spoke on Enlarged Prostate.

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A meeting of the branch was held on 21-4-56. Dr. V. Enok presided. Fifty members were present. Office-bearers for the 11th Madras State Medical Conference were elected. The Reception Committee membership fee was fixed at Rs. 10/- and that of visitors at Rs. 5/- only. Dr. M. V. Chari gave a talk on Recent Advances in Pediatrics.

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A meeting was held on 24-3-56. Fifty members were present. Dr. V. Enok presided. Dr. C. S. Sadadivam spoke of Government Tuberculosis Sanatorium. Tambaram spoke on "Management of Empyema".

UTTAR PRADESH STATE BRANCH—A report of the activities of the branch for the year 1955 shows that Apka Swasthya was given a token help of Rs. 200/- during the year. This paper is progressing steadily. In addition to 500 copies subscribed by the Government of India, the U.P. Government has subscribed 800 plus 834 plus 150 copies per month, the Jammu and Kashmir Government 100 copies and Patna District Board 25

copies. The branch spent Rs. 1,000/- towards flood, out of this, the central office contributed Rs. 500/- only.

VIJAYAWADA BRANCH—A special meeting of the branch was held on 17-4-56 with Dr. C. Suryanarayanamurti in the chair. Dr. P. Tirumala Rao of K. G. Hospital, Vijagapatnam spoke on Emergencies in Pediatrics and showed a few slides.

VISAKHAPATNAM BRANCH—The annual general body meeting of the branch was held on 24-3-56 with Dr. M. V. Krishna Rao in the chair. The annual oration was given by Dr. M. Krishnamurthy, Professor of Surgery, Andhra Medical College, on "Enuresis in children".

WEST GODAVARY BRANCH—The annual conference of the branch was held on 1-4-56, with Dr. V. V. G. Tilak in the chair. Fifty doctors were present. Dr. K. Somayya, Dy. Director of Public Health, Andhra State, inaugurated the conference. Dr. V. Rama Doss opened the scientists' portraits gallery. The annual report was adopted. In the scientific section 14 doctors gave scientific lectures during the year. Office-bearers were elected with Dr. V. V. G. Tilak as president, Dr. R. Narasinha Raju as hony. general secretary and Dr. P. S. Kameswara Rao, Dr. C. Ranga Rao and Dr. K. Gunneswara Rao as hony. joint secretaries. A symposium was held on tuberculosis.

XXXIII ALL INDIA MEDICAL CONFERENCE. 1956.

The Scientific Transaction Committee of the XXXIII All India Medical Conference which will be held in Trivandrum in December has decided to hold symposia on the following subjects:

Medicine—Coronary Heart Disease; Surgery—Oral Cancer; Social & Preventive Medicine—School Health Services; Obstetrics & Gynaecology—Haemorrhages.

The time allotted for each speaker is ten minutes.

Those who desire to read papers at the Symposia are requested to submit the title of their subjects to the Secretary, Scientific Transaction Committee, Trivandrum, before the 31st July, 1956. A summary of the paper may kindly be sent before the 15th September, 1956. Arrangements may also be made for longer papers or talks on important subjects of general interest if the abstract of the paper is received not later than 1st October, 1956.

JOURNAL OF THE INDIAN MEDICAL ASSOCIATION

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